ARCHIVES OF OPHTHALMOLOGY.

GLAUCOMA AND AFFECTIONS OF THE OPTIC NERVE.

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(*With Twenty-one Wood-cuts.*)

IN his extensive treatise on glaucoma[1] von Graefe laid stress upon the differences between that disease and certain other forms of visual disturbance, which he designated "amaurosis, accompanied by excavation of the optic papilla." I shall quote some of his statements bearing upon this subject, as I consider them as still entirely true: "In some cases which had been diagnosed as glaucoma after an ophthalmoscopic examination only there were no external indications of the glaucomatous condition to be found. The alteration in the nerve itself constituted the only evidence of the disease." "In the absence of other symptoms of increased tension we are not justified in regarding such affections of the optic nerve as a direct result of intraocular pressure. It seems to me that it is absolutely necessary, if we are to understand these cases, that they should be removed from the category of glaucomatous diseases, and studied under the above-mentioned or some other title." "Although from my own experience I have learned that there is much uncertainty in studying these matters, yet I cannot too strongly insist that in the present state of our knowledge the *optic-nerve affection alone is not sufficient to warrant a diagnosis of glaucoma; in many instances it has a pathogenesis entirely distinct from the latter disease.*"

[1] *v. Graefe's Archiv*, 1857, Bd. iii., 2.

Unfortunately, however, the study of these affections has not proceeded upon the lines laid down by von Graefe, but under a new name has been carried on in quite another direction with anything but fruitful results. In 1861 (with Donders' approval) von Haffmann's [1] article appeared. In it typical *glaucoma simplex* is described, and the inflammatory manifestations of glaucoma are considered as a complication of the disease. Nothing is said about amaurosis associated with excavation of the disk.[2] "Experience has taught us that in every instance where the characteristic form of the excavation is present, *and it stretches to the margin of the papilla* even when no increased tension has been discovered, the latter must be considered, if not as the morbid condition itself, at least as the chief sign of it. It must be remembered that many symptoms of increased tension may be absent. The skilled finger will, however, immediately and unerringly discover the presence of increased tension. It may be present in only a slight degree and have very slowly developed." "The difference between von Graefe's observations and our own is that we believe increase of tension, more or less marked, always accompanies the typical excavation just referred to. We might second von Graefe's contention had we been able to find a case where such a papillary excavation was present without increased tension." [3]

It is not too much to say that von Graefe may be trusted to have examined with "skilled finger" his cases of amaurosis with optic-nerve excavation. In the same way it is now conceded by every one that cases of excavation reaching to the margin of the disk are not uncommon where the skilled touch has been unable to detect any trace of increased tension. The candid, unprejudiced observer must admit that increase of intraocular pressure, however important its pathological significance, must be carefully considered in the light of other diagnostic evidence. Augmented tension and glaucoma are by no means identical conditions. I have, for

[1] *L. c.*, Bd. viii., 2, S. 124.
[2] *v. Graefe's Archiv*, Bd. viii., 2, S. 147.
[3] *L. c.*, Bd. viii., 2, S. 150.

example, seen the former occur temporarily in severe attacks of iritis, and yet, although observed for years afterwards, the eye presented no sign whatever of glaucoma. In many such cases I have been unable to satisfy myself that the increase of tension had not been produced by spasmodic contraction of the ocular muscles when the eyeball was pressed upon. Moreover, I have observed unilateral plus tension as a temporary manifestation in eyes unaffected by either inflammation or glaucoma. On the other hand, increase of tension may, *just at the time of examination*, be doubtful or altogether absent in cases where glaucoma was certainly present. It often happens that a satisfactory estimation of the ocular tension is rendered unsatisfactory owing to spasm of the orbicularis and the other external muscles, having been produced by the ordinary tactile examination. There is no such thing as a standard of hardness by which the tension of all eyes can be determined. The sense of resistance conveyed to the finger depends not only upon the intraocular pressure, but upon the rigidity of the sclera, which varies not only in individuals but even in different parts of the same eye. This is especially true of myopes, where in the posterior half of the globe there is more or less stretching and thinning of the sclerotic. There are eyes that are physiologically hard and eyes that are physiologically soft. The most reliable method of all is to compare the hardness of the two eyes, but even then it is often difficult in abnormal cases to decide whether there is increase of tension or not. Indeed, the diagnostic value of this "objective" examination is often affected by subjective factors. Whenever an observer decides that there *ought to be* increased tension, his "practised finger" will surely feel it. And then follows the determination of the particular degree of tension with a T + 1, + 2 or + 3! Does any one imagine that two independent examiners would be sure to report T + 2 in the same case? The results are very little better with the tonometer and with manometric measurements.[1] In my

[1] See, for example, *v. Graefe's Archiv*, Bd. xxix., 2, S. 11, and Bd. xxxi., 1, S. 105. In the former place atropine is found by manometric measurement to *increase*, in the latter to *diminish*, the intraocular pressure!

opinion the best plan to determine the ocular resistance is that of Coccius—to press directly upon the sclera. Patients who make this method of examination impossible by spasmodically squeezing their lids together will do the same thing if you attempt to touch their eyelids.

The weakest evidence of all is the "typical" excavation. Every excavation which reached the papillary margin was without more ado set down as glaucomatous, and every case which von Graefe was careful to specially designate as amaurosis with optic-nerve excavation, was labelled glaucoma simplex. Of course in this category of von Graefe's were included not only cases of true glaucoma, but also others whose excavation-sign had quite another significance. But that was just the task which he had set himself —how to distinguish, by means of further observation, the one class from the other. However, the "typical" excavation theory carried the day; everybody was converted to the new gospel, and it is not an over-statement to say that they then became a prey to tactile hallucinations, inasmuch as no one now denies that deep papillary excavations, as well as those that extend to the border of the disk, are not infrequently present without the least sign of increased tension.

If we inquire into the conditions under which such excavations occur without glaucoma, we must begin with a study of the physiological excavation. Our knowledge of the physiological leads us to an appreciation of the pathological. That the optic-nerve fibres may reach the plane of the retina they must necessarily make a rectangular bend; consequently there is always a small pit in the centre of the papilla, which is usually of a lighter color than the rest of the disk, and often presents the form of a funnel. Its width and depth vary greatly; it may involve most of the papillary surface, and so be wide and shallow, or it may be quite small and deep.

To draw the line somewhere, I might define the term physiological excavation to be one which includes more than one third of the disk surface, and whose floor is formed by the lamina cribrosa. If one discovers such

an excavation in one nerve, he may confidently expect to find a similar pit in the other. There may be slight variations in their breadth and depth, especially in those large excavations that involve more than half the surface of the papilla, and approach its margin. In several of these instances of large physiological excavation I have been able to trace a hereditary transmission. I do not believe that they are associated with any particular refractive condition, hypermatropic or other. Frequently the bottom of the excavation (the lamina cribrosa) lies considerably behind the plane of the choroid.

When a physiological cavity includes more than one half the disk surface and the lamina cribrosa lies deeper than usual, the fibres that issue from the middle of the nerve trunk hug the walls of the excavation, and do not make their rectangular *bend* until they reach the papillary margin. Consequently the nerve fibres that supply the medial half of the retina constitute the medial border of the cavity, while those fibres that go to the temporal part of the retinal periphery form its upper and lower margins because the temporal border is reserved for the macular fibres. This is also true of the blood-vessels. The temporal border of the physiological excavation in consequence of this presents, in most cases, a lighter coloration and (as may be seen by observing the small vessels that run towards the macular region) a less steep declivity. The medial border of the physiological cup, on account of its rich vascular supply, is of a deeper reddish-gray color, and in consequence of the above-described disposition of its nerve fibres usually assumes a semilunar shape. In the case of a large excavation its margin is almost identical with the edge of the disk, since the layer of nerve fibres at the retinal border is only 0.2 *mm* thick, about the same thickness as the layer which lies between the excavation margin and the border of the sclerotic. If we consider the diameter of the optic nerve to be 1.5 *mm*, then the thickness of the nervous layer at the papillary border is about one eighth of the whole. In the ophthalmoscopic picture it looks as if the papillary margin and border of the excavation were one and the same. This

is not always true of its entire circumference, but it is very often the case with the upper border of the excavation. The larger retinal vessels project at the border of the physiological pit, and then bend abruptly like a hook. While they are closely applied to the wall of the pit, they are visible either in foreshortened perspective, or are not seen at all. Inasmuch as in an ophthalmoscopic examination the observer is confined to the view obtained in a straight line only, it can happen that the overhanging medial border of the excavation may hide, or almost hide, the point at which the retinal vessels are given off.

All these conditions obtain in glaucomatous excavations, and for the same reason. When, as in elderly persons, the retina loses its lustre and transparency, this resemblance to pressure-cupping becomes still more marked. Indeed cases have come under my observation where solely on account of such excavations eminent oculists have subjected their patients to iridectomy, and have even produced in them traumatic cataract, while certain others, whose physiological excavations were stated to call for urgent operation, only saved themselves by immediate flight. These latter, remaining under my observation for years, continued to have normal visual acuteness, and to retain an unrestricted visual field.

When there is joined to a pre-existing physiological excavation an optic nerve atrophy, the difficulties of diagnosis are greatly increased. Müller[1] has pointed out that in the latter case connective-tissue remains are visible on the lamina cribrosa posterior to the retinal plane—a condition which he describes as atrophic excavation. I have pictured such a case in my "Lectures on the Ophthalmoscope."[2]

The accompanying wood-cut, Fig. 1, is an exact drawing from the preparation itself. The retina (*R*) is somewhat detached from the choroid (*Ch*). *V.r.* is a transverse section through the retinal vessels, and *V.c.* indicates the central artery and vein.

[1] *v. Graefe's Archiv.*, Bd. iii., 1, S. 92.
[2] Berlin, 1864.

Fig. 1 is an illustration of how in atrophic conditions of the nerve the retinal vessels may disappear at the margin of an excavation. As opposed to a diagnosis of glaucoma in such an instance is the position of the lamina cribrosa—reaching to the choroidal plane; in an excavation due to pressure it lies considerably deeper than the latter. It follows from this that the larger the pre-existing physiological excavation the closer is its resemblance to the glaucomatous cup should the former become the seat of atrophy.

The lighter color of an atrophic optic nerve has an important bearing upon this question of pressure-excavation. Whether the atrophy is caused by the pressure-excavation or whether it has simply developed in a previously normal ex-

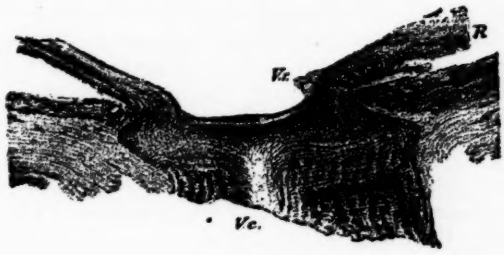

FIG. 1.

cavation can hardly be settled by the ophthalmoscope alone, for in the latter case the marginal line of the excavation may coincide exactly with the scleral border of the nerve and so present a "typical" picture of an excavation due to intra-ocular pressure, and yet be actually a result of optic atrophy without glaucoma.

This confusion of optic-nerve affections with simple glaucoma is as old as the word itself. As an example of this see "Contributions to the Study of Glaucoma,"[1] in which a case of "glaucoma simplex" is described that produced blindness in thirteen weeks. The state of the visual field, but still more the clinical history, are opposed to the idea of glaucoma; the latter disease does not go on to complete loss

[1] *L. c.*, Bd. viii., 2, S. 145.

of sight in so short a time, although some optic-nerve diseases may. So far as plus tension is concerned, there is no difficulty in discovering that, if one only has sufficient faith! I am convinced that all cases described as glaucoma simplex fulminans are really instances of atrophic degeneration of the optic nerve occurring with physiological cupping of the disk.

If this be true there is no such thing as a *typical* excavation due to intraocular pressure, and when the character of the excavation is in doubt (especially in bilateral cases) it is not to be determined by testing the tension merely; all the other symptoms of glaucoma must be considered. The so called "glaucoma simplex" may thus be divided into two groups: first, true glaucoma, unaccompanied by marked inflammatory signs; and, second, a diseased optic nerve with a pre-existing physiological excavation. In what respect do these two groups differ from each other? I have already (in 1877[1]) drawn attention to the importance of this question, but shall now proceed to consider it more closely.

If one find in the papilla of one eye a marked excavation while the disk surface of the other is flat, it is certainly a case of glaucoma, because that is the only disease capable of excavating the disk. When, however, it is discovered in both eyes the diagnosis is more difficult, chiefly because optic-nerve atrophy may be the result of not one but of many quite different affections. Von Graefe has already cautioned us against the loose application of the term "optic-nerve atrophy" still in vogue among us, and has pointed out the importance of differentiating between atrophic degenerations and primary atrophy of the nerve fibres. Secondary atrophic degeneration may result from all those diseases which interfere with light-conduction in the retina or optic nerve, *e. g.* choroiditis, retinal hemorrhage, neuroretinitis, retrobulbar neuritis, retinal embolism, etc.

Still easier is it to mistake a physiological excavation for glaucoma when the former is associated with certain forms of primary atrophy. The commonest variety, spinal atrophy,

[1] "Ueber Glaucom." Clinical lectures published by R. Volkmann, No. 124, p. 1043.

presents such characteristic signs that it is not likely to mislead, but the more uncommon form, that unconnected with spinal disease, may very easily be mistaken for simple glaucoma in the presence of a physiological excavation of the papilla. It is consequently advisable to watch the course of such cases, particularly in eyes that present no excavation of the disk. Sometimes their progress, just as in glaucoma simplex, is a very chronic one. I will, as an example, give the following history of such a case with a physiological excavation, which might very easily have been mistaken for glaucoma simplex:

CASE 1.—R. S. æt. 76, in 1876 had u. o. V = $\frac{6}{9}$. Fundus normal then and after repeated examinations in 1878 and 1880. In April, 1881, V = $\frac{5}{12}$ — $\frac{6}{9}$. O D a light gray. In October, 1885, V L=fingers with difficulty; a defect in the lower visual field; light reaction normal but O D white. V R = $\frac{5}{18}$; O D almost as white as L. In June, 1886, V L = hand movements. The atrophy had produced retraction of the papillary surface and a slight incurvation of the vessels was noticed at its border. Further loss of vision followed until death in 1888. Period from the commencement of the optic-nerve disease until complete blindness set in from six to seven years.

In another similar case, that ran its course to total blindness in eleven years, glaucoma was excluded by the early discovery of disturbed color-sense, while in the following case the mistake was actually made.

CASE 2.—von B., in 1882 had V = $\frac{1}{2}$ – 1. Apart from some peripheral lenticular opacities his eyes were normal. A small pit in the centre of the O D. In June, 1884, a slight discoloration of the papilla was noticeable. V R and L = $\frac{6}{9}$. December, 1885, left upper hemiopic defect in F of V following a slight attack of apoplexy. Both papillæ gray and showing a slight incurvation of the vessels at the medial border of the nerve. December, 1886, V R = $\frac{1}{3}$ – $\frac{1}{2}$, V L = $\frac{1}{4}$. This deterioration of V in the left eye showed itself after a sclerotomy had been perpetrated upon it, resulting in astigmatism visible by the keratoscope. In November, 1888, V R = $\frac{5}{12}$; V L = $\frac{5}{36}$ – $\frac{5}{120}$; O D as before, grayish-white. Patient died in 1889 of mental disease.

It is worthy of notice that the senile decoloration of the disk was so pronounced in this case that it was mistaken for an excavation; that there was no atrophic degeneration present is shown by the fact that for four years there was little or no loss of vision, except that due to the operation.

I have, of course, seen cases in which atrophic degeneration has been mistaken for an excavation, and by taking a step farther, one might, as C. Stedman Bull[1] has done, designate cases without excavation glaucoma simplex simply on the ground of deteriorated vision and a contracted field.

Finally, that atrophic degeneration of the nerve does not necessarily lead to blindness, but may remain stationary

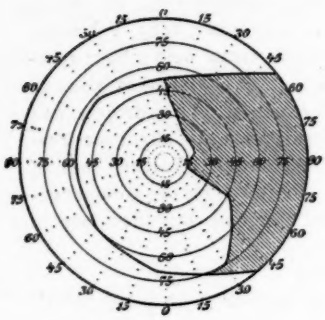

FIG. 2.

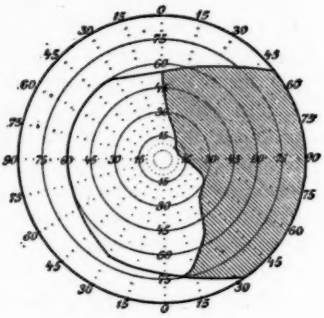

FIG. 3.

with an extensive defect in the visual field, is proved by the following history.

CASE 3.—Mr. M., æt. 50, consulted me about his presbyopia in December, 1874. Both eyes emmetropic and V = $\frac{10}{10}$. July, 1875, sight unchanged; V L = $\frac{10}{30}$; outwards a large scotoma, color-sense and pupillary reaction normal; O D somewhat "off" color. The vision in this eye grew gradually worse until, at the end of 1876, complete blindness resulted. The R E continued normal until April, 1879, when a small outward scotoma and a slight discoloration of the disk were discovered. Fig. 2 shows the F of V in May, 1879. Patient having had syphilis 20–25 years before, mercurial inunction was employed, with complete

[1] *N. Y. Med. Jour.*, Aug. 10, 1889.

arrest of the disease. When last examined, January, 1888: V R = $\frac{8}{9}$; the F of V was as in Fig. 3; and the O D slightly paler than normal.

The inunction treatment may have saved this patient, but almost every case of atrophic degeneration of the opticus free from spinal disease has been treated by me in this way without benefit. It is generally conceded that in the spinal form it is also valueless.

Since all these cases were complicated by the presence of physiological excavation they were doubtless difficult to diagnose from simple glaucoma. That I may lay additional stress upon the importance of diagnosis I report the follow-

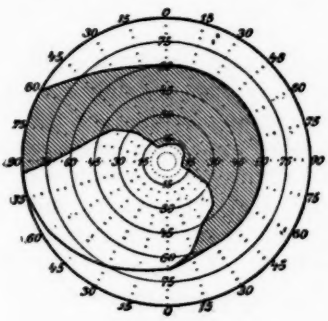

FIG. 4.

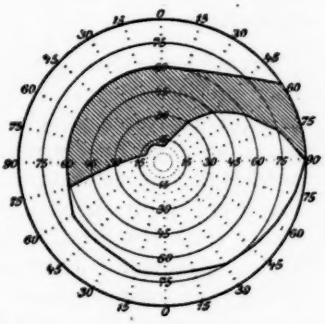

FIG. 5.

ing case of "typical glaucomatous excavation" *without* glaucoma, that ran its slow course to final blindness.

CASE 4.—Miss G., æt. 66, consulted me because some one had frightened her about her "excavation." V R + 1.25 = $\frac{6}{12}$; V L + 1.25 = $\frac{6}{9}$. Deep bilateral excavations reaching to the margin, but F of V uncontracted. Cornea clear; pupil normal; T + ? No halos or cloudy vision. No trace of glaucoma or excuse for iridectomy. In May, 1888, patient returned, after consulting another oculist, with vision decidedly worse. L V + 1.25 = $\frac{5}{24}$; scotoma up and in; V R + 1.25 = $\frac{5}{12}$; also a defect in the F of V (Figs. 4 and 5). Color-sense, cornea, and pupil normal. Papilla pale. Although I saw no reason to modify my previous diagnosis, I felt obliged to do an iridectomy. The

case was growing worse, and as the patient had been urged to undergo the operation the increasing blindness would naturally be attributed by every future observer to my neglect. However, vision gradually sank until about a year after the iridectomy. V L + 1.25 = $\frac{1}{5}$; V R + 1.25 = $\frac{1}{5}$. In November, 1889, the question of glaucoma was decided, so far as the left eye was concerned, by numerous retinal hemorrhages. V = fingers at 20 *cm*; V R + 1.25 = $\frac{5}{24}$. The scotoma gradually approached the fixation point. In the right eye marked tortuosity of the small retinal vessels. This is plainly marked at the bottom of the excavation —an observation I have made in similar cases of physiological excavation accompanied by atrophy, and consequently not to be regarded as characteristic of glaucoma. The F of V in the right eye contracted still more, and vision was finally lost from retinal hemorrhage.

From the beginning the term glaucoma simplex has not been precisely defined.[1] "Every oculist has certainly seen cases of excavated papillæ with increasing hardness of the globe, etc." "The increased tension from fluid pressure persists and becomes so great as to bring about atrophy of the nerve at the papilla, contraction of the arteries, and finally total blindness." This is said to be a picture of typical glaucoma without complication. I deny it. I also doubt the statement made on pages 151 and 155 that in simple glaucoma the eye may assume a stony hardness. On page 155 it is said that "inflammatory symptoms, as evidenced by hemorrhage, pain, and cloudiness of the media, often set in during the early stage of the disease." That is to say simple and inflammatory glaucoma may exist in the same eye.

This authority seeks to explain (page 150) von Graefe's initial stage of simple glaucoma by regarding it as the glaucoma itself. What meaning is attached to the term glaucoma simplex? Possibly the intermediate stages when the eye is absolutely normal and without glaucomatous symptoms, or does simple glaucoma begin with an initial stage of cloudy vision, halos, etc.? The term "initial stage" is an unfortunate one, as slight attacks are not necessarily

[1] *v. Graefe's Arch.*, Bd. viii., 2, S. 143.

the forerunner of more severe ones; gradually and without the latter the eye may slowly advance to blindness.

These slight passing attacks of glaucoma controvert the retention theory of the disease. How can one reconcile with this theory the fact that the symptoms set in so abruptly and disappear so quickly? In what way does the filtration angle become so quickly blocked up and as suddenly restored? Swelling of the ciliary processes has been held responsible for it. That inflammatory alterations may be discovered in eyes enucleated on account of painful, severe, and long-continued inflammation is self-evident. The ciliary processes in these cases may be atrophied or they may be swollen, but it does not necessarily follow that glaucoma begins with ciliary swelling. One of the few instances in which a recent case of glaucoma was examined anatomically is reported by Birnbacher.[1] The eye was examined seven days after the disease—acute inflammatory glaucoma—showed itself. The ciliary processes were greatly developed, but not to a greater extent than is often seen in normal eyes. There was neither swelling nor softening of the ciliary tissues; the blood-vessels presented no sign of parietal changes or of congestion.

Finally the greatest individual differences exist as to size between ciliary bodies. Fuchs[2] has described a case of glaucoma operated upon seven years before and with good results, in which after the death of the patient an uncommonly well developed ciliary body was found. Inasmuch as the eye had been free of glaucomatous symptoms for seven years, the extreme ciliary development cannot be regarded as the cause of the original disease. Otherwise one could hardly be asked to believe that with the cause continually active the effect should be entirely removed.

An obstruction at the filtration angle produced by ciliary enlargement would, in the beginning at least, result in an increase of aqueous and a deepening of the anterior chamber—a state of things which has never been observed. As everybody knows, the shallow anterior chamber of glaucoma

[1] "Beitrag zur Anatomie des Glaucoma acutum." Graz, 1890.
[2] *v. Graefe's Archiv*, Bd. xxx., 3, S. 123.

is produced by a crowding forward of the crystalline and its attachments consequent upon an increase in the volume of the vitreous. In Birnbacher's report not only is this stated to be the case, but owing to tension upon the zonula the ciliary processes were dragged forward to the posterior surface of the iris, and in places came into actual contact with it. This, however, is not the cause, but simply the effect of increased intraocular pressure. The occurrence of glaucoma in aphakic eyes also bears witness against the retention theory. Of course, glaucoma rarely follows aphakia, but then aphakic eyes are much less numerous than those that are supplied with lenses. Moreover, a deep anterior chamber with aphakia is hardly compatible with the idea of an obstructed filtration angle due to swelling of the ciliary processes.

Probably the chief and most frequent symptom of glaucoma is misty vision and the appearance of halos. These symptoms must not be *suggested* to the patient. Almost every one with weak sight complains of cloudy vision, and even "rainbows" may after a number of observations be seen, which are, however, often merely sensory hallucinations. Yet these statements must never be lightly regarded, especially when glaucoma is already present in one eye. That true halo-vision does not always indicate glaucoma is well known. The thin film of mucus that sometimes overspreads the cornea in cases of chronic conjunctivitis gives rise to these symptoms.

In examining cases of glaucoma in which halos have been seen, the observer will notice, along with conjunctival hyperæmia and increased tension, a slight cloudiness of the cornea. It is consequently a true diffractive effect, such as one obtains by looking through a misty window-pane, or by gazing at a light through a glass sprinkled with lycopodium powder. It is only in cases of slight cloudiness of the cornea that this appearance is seen; when the opacity is so marked as to interfere with an ophthalmoscopic examination, it is not perceived by the patient. That this symptom is rarely noticed by the patient is due to the fact that, as a rule, patients are not acute observers. For example, *in mild*

attacks of iritis with slight deterioration (less than about $\frac{1}{8}$) of vision, halo-vision may readily be observed if the sound eye be covered and the patient be told to look at a lamp that has been turned down low. Not infrequently patients tell me of this appearance, and in cases like the foregoing it can readily be induced. The same is true of glaucoma, and if we regard the cloudy cornea in iritis as an inflammatory change, we must attach a like significance to it in glaucoma. In both cases it is an inflammatory œdema of the cornea. In this, as in other conditions, there are marked individual differences. For instance, it not infrequently happens that with slight hyperæmia of the conjunctiva, without pain, with a moderate degree of tension, and with an active pupil, the cornea may be so cloudy that the fundus is barely visible.

Finally, there is nothing characteristic about the corneal opacity; it is precisely the same in kind as that which accompanies iritis and irido-choroiditis. In connection with this occurs the question of a cloudy aqueous in glaucoma; but how can one distinguish through a cloudy cornea a turbid aqueous humor? The assertion that sometimes immediately after the escape of aqueous following iridectomy the pupil appears clear, proves nothing unless the cornea is examined by focal illumination, which in many cases is alone capable of indicating a slight but evident haze of the cornea. Furthermore, it is a rule that the corneal cloudiness only gradually disappears after iridectomy. The same thing may be said of the oft-quoted vitreous haziness. Glaucoma and a cloudy vitreous are not of necessity mutually exclusive, but the latter can only be demonstrated through a transparent cornea.

Another very important sign is the condition of the pupil. Glaucoma may progress to complete loss of sight without interference with the mobility of the iris. In many cases of unilateral blindness from glaucoma the direct reaction to light, of course, fails, but the convergent consensual-contraction remains.

Of the greatest diagnostic value is the dilatation of the pupil in glaucoma with inflammatory symptoms. In a glaucomatous attack, if the pupil be enlarged, we shall also

find a cloudy cornea and increased tension, if the latter has not already passed away.

It rarely happens that the pupil is enlarged and at the same time round and perfectly movable. When one eye is completely blinded by glaucoma, such a condition might be induced if the other normal eye were accidentally covered. Avoiding this source of error I have, both in unilateral blindness from glaucoma, as well as in the earlier stages (with slight inflammatory symptoms), seldom found the pupil even moderately dilated and at the same time round and completely movable.

In most cases this enlargement of the pupil may be regarded merely as a ciliary paralysis from pressure, and as in other instances of this sort may be temporary or permanent. That the ciliary nerves are very sensitive to pressure is proved by the occurrence of *mydriasis traumatica*, which may follow contusions of the eye that leave behind them no other bad effect. It would be most unfortunate if the other motor nerves were as subject to traumatic paresis as the ciliary. In glaucoma paralysis of the ciliary nerves may be produced by one attack of sharply increased tension, but such a paralysis is not always complete. Sometimes the pupil is dilated in one direction only—most frequently upwards,—while in the remainder of the circumference the pupil still reacts to light, showing that some of the nerve fibres still retain their conducting power. Whenever we find the pupil fixed and widely dilated we may feel sure that the eye has been subjected to severe attacks of increased tension. Of course the presence of an inactive pupil does not necessarily imply glaucoma, but I have seen cases of atrophic degeneration of the optic nerve with immobility of the iris due to disease of the nerve centres diagnosed as glaucoma.

What has been said of the dilated pupil applies equally to the shallow anterior chamber. It is produced by a sudden increase of intraocular pressure accompanied by inflammatory symptoms due to enlargement of the vitreous body. The anterior space is again restored when the abnormal tension disappears. The diagnostic value of this sign is small.

Another symptom directly arising from this condition of increased tension is arterial pulsation, whose importance in the diagnosis of glaucoma von Graefe has already drawn attention to. Of course I refer only to the pathological form of arterial pulsation. A physiological increase of pressure in the retinal arteries follows the cardiac diastole, but its effects are not visible; only under certain pathological conditions, such as in affections of the heart and large vessels, Basedow's disease,[1] etc., can the systolic expansion and movement of the arteries be seen.

The arterial pulse, which in glaucoma can always be induced by pressure on the globe, is a decidedly abnormal occurrence. It may be recognized by the fact that *during* the heart's diastole the central artery is in places quite empty of blood. This arterial pulsation is the result of a disproportion between the intravascular pressure in the arteria centralis and the vitreous pressure. During the diastole the pressure of the vitreous preponderates and compresses the arterial walls at their weakest point—just where the vessel leaves the nerve trunk and makes a rectangular bend. That part of the artery thus emptied of blood commonly reaches to the margin of the disk, rarely past it. During the systole the blood pressure is in excess and the emptied vessel again becomes filled.

Usually the cause of the phenomenon lies in increased intraocular tension, but this is not always the case, since whatever reduces arterial tension may produce the same result. For instance, von Graefe[2] observed two examples of arterial pulsation in orbital tumors accompanied by neuroretinitis. It is possible that the tumor may have exerted on the eye an effect similar to finger pressure, or it may have compressed the ophthalmic artery and in this way reduced the intravascular tension. In a third case, of neuritis, it was probably produced by the space-reduction within the scleral ring—a result of the neuritis itself. Exactly the same condition may be induced by a general reduction of

[1] Quincke, *Berlin. klin. Wochenschr.*, 1886, No. 34, and 1870, No 21. Also O. Becker, *v. Graefe's Archiv*, Bd. xviii., 1, S. 206, and *Wiener med. Wochenschr.*, 1875.

[2] *v. Graefe's Arch.*, Bd. xii., 2, S. 131.

the blood-pressure as an expression of cardiac weakness. In a case of syncope B. Wordsworth[1] observed the arterial pulse set in immediately before the collapse of the patient.

Thus pulsation of the central artery can be produced even when the intravascular tension is normal if the intraocular tension be raised; it is also occasioned by the normal ocular tension when the arterial pressure sinks sufficiently low. It follows from this that in glaucoma the occurrence of arterial pulsation does not necessarily imply a marked increase of intraocular pressure. *I consider arterial pulsation alone to be insufficient evidence of glaucoma.*

Doubtless if a clear view of the fundus were not so frequently prevented by a cloudy cornea we would be able to discover in glaucoma more cases of arterial pulsation. Of greater importance is it to detect it in glaucoma without concomitant inflammatory conditions, even without the usual results of increased tension, such as dilatation and immobility of the pupil. I have repeatedly observed it when the pupil was normal and active, when the vision was normal, and when the tension was slightly increased or doubtful. I have often seen the central artery pulsate in such cases after simple movement of the eye.

That one can induce arterial pulsations by pressure of the finger upon the normal eye has been observed by von Graefe[2] from experiments on his own eye. "It is extremely interesting but somewhat dangerous to observe the arterial pulse in one's own eye after one has induced 'Purkinje's figures.' It seems to me that one can in this way follow the pulsation quite a distance from the blind spot. The very strong pressure, however, produces such severe and lasting symptoms that I would not advise any one to try it on his own eyes."

Of special significance is arterial pulsation in connection with retinal hemorrhage following iridectomy. When the anterior chamber is emptied of its contents the intraocular pressure instantly falls and blood rushes into the retinal vessels. As Cohnheim[3] has shown, this is a condition that

[1] Ophth. Hosp. Reports, vol. iv., p. 111.
[2] *v. Graefe's Arch.*, Bd. i., 1, 8, 387–688.
[3] "Untersuchungen aus dem embolischen Process," 1872, S. 39.

favors hemorrhage; he has observed that the capillaries, for a time shut off from the circulation, are liable to rupture when the blood stream is again turned into them.

Of course arterial pulsation is not a necessary forerunner of these bleedings. I have seen them take place in cases that just before the operation showed no sign of pulsation. This is also true of bleeding into the anterior chamber. It often occurs along with retinal hemorrhages, but the latter is such a common accident that it is not easy to establish a causal relation between them. It is worthy of remark that hemorrhage from both iris and retina may follow iridectomy for glaucoma in cases presenting few inflammatory symptoms.

But the most important alteration that accompanies glaucoma is that which the optic nerve suffers. It must, however, be remembered that there is but one way in which a correct estimate of the clinical meaning to be attached to a pressure-cup can be reached, and that is by comparing it with an earlier picture of the papilla. *Furthermore, I may state that glaucoma may proceed to final blindness without the formation of an excavation.* The following is a case in point.

Case 5.—Mrs. F., æt. 54, came under treatment the beginning of December, 1878. Two years before said to have had severe inflammation of *right* eye with headache. Three months ago another painful inflammatory attack in the same eye, which resulted, after about eight days, in complete blindness. The *left* eye was said to have had good vision until eight days ago, when she had a severe attack of inflammation accompanied by nausea and vomiting. Vision is almost entirely destroyed. Only inwards and upwards are hand movements still perceived. There is a slight degree of tension in both eyes—more in the right than in the left. Bilateral pericorneal injection; cornea cloudy, the central area most affected; anterior chamber not encroached upon; red reflex indistinct; eserine produces but slight effect.

December 3, 1878 (under chloroform) T ?; sclerotomy in both eyes. December 13th, right cornea transparent; optic nerve excavated; no retinal hemorrhages; *left* cornea transparent; *no excavation of papilla, which appeared quite normal.* Vision finally reduced to perception of light and the uncertain discernment of large objects in eccentric portions of the field.

It is worthy of note that a *single* attack of glaucoma can induce blindness in an otherwise healthy eye. When the patient above referred to applied for treatment, eight days after the attack, the increase of tension was fast disappearing. The optic nerve, visible after the operation, seemed quite normal, and, although later on atrophic degeneration showed itself, there was no excavation.

In addition to this, large defects in the field of vision may be developed without excavation.

Case 6.—Mrs. L., æt. 36. January 23, 1883: *Right*, sharp attack of inflammatory glaucoma for past eight days; cloudiness of cornea and mydriasis; V = hand movements only; defect of medial half of field. Iridectomy, January 24th. Evening of January 25th, *left*, slightly cloudy cornea and dilated pupil. In the night a severe attack. January 26th, iridectomy. February 13th: R V = fingers at 1 metre. O D not excavated but lighter in color. November 17th: V R = $\frac{1}{18}$; O D not excavated but white; V L + 1 = $\frac{1}{8}$; O D normal.

Case 7.—Florentine L., æt., 49. March 27, 1882: Glaucoma in *right* since November. V = fingers at 1 *m*. Contracted F of V; mydriasis and haze of cornea; fundus not visible; + T?; slight effects from eserine; V L + 4 = $\frac{6}{18}$; previous convergent strabismus. March 28, 1883: *Right*, iridectomy. April 7th: Still some blood in anterior chamber; no excavation; O D somewhat paler than left; some small retinal hemorrhages at the equator outwards; V + 3 = $\frac{1}{12}$ + 6. No. 0.3 slowly; scotoma above and in.

Fig. 6 is the perimeter chart, measured March 27th. It is not an exact one, as might be expected from the low vision. Fig. 7 shows the F of V projected on a flat surface; the medial scotoma corresponds in form to the perimetric measurement; the temporal half of the field, on the other hand, was normal.

In all these cases, if there had been a physiological cup, a marked excavation from pressure would have been seen, but this would not have been the cause of the blindness, inasmuch as the latter resulted from pressure-paresis of the retina or the optic nerve. At first the papilla looked quite

normal, and it was only later on that atrophy showed itself as a result of the blindness, just as, for instance, we find it following destruction of the opticus fibres from hemorrhage into the retina. Of course such a paralysis due to pressure presupposes a sharp increase of the intraocular tension which only occurs in severe attacks of glaucoma. In those cases that pursue a chronic course, the formation of an excavation is a gradual process, and when a physiological pit already exists it affects like it the centre of the disk. I have seen cases, in which there was no physiological pit, after the arrest of the disease by iridectomy, present an ex-

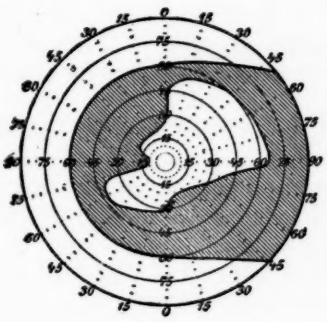

FIG. 6.

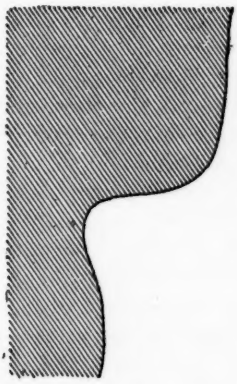

FIG. 7.

cavation that, in so far as it did not extend to the papillary margin, closely resembled the normal condition. In the earlier stages of the pressure-cup the atrophic discoloration is commonly absent, but when the glaucoma is of long standing it can always be seen. *The ophthalmoscopic picture of a physiological cup may be produced by glaucoma, just as extensive normal excavations with discoloration of the disk cannot be distinguished from excavations due to intraocular pressure.* Small physiological excavations (which affect about one third of the disk surface) are quite common, and the larger the normal cup the sooner it enlarges under the influence of pressure and reaches the border of the papilla. All this once

more teaches us not to allow the diagnosis of glaucoma to rest upon a simple symptom, but to take into consideration every clinical feature. The length of time occupied by glaucoma in the excavation of a level disk depends upon a variety of circumstances. It is not uncommon to run across cases that have persisted for years, having a history of attacks accompanied by cloudy vision and halos, until a more severe attack than usual called for an operation, and yet no pressure excavation resulted. The occurrence of this sign is evidently governed by the amount of the intraocular pressure, the duration and frequency of the several attacks, and the power of resistance inherent in the nervous tissue itself. Finally, a disk with even a small excavation sooner presents the appearance of having been excavated by pressure than does a flat papilla with little or no central pitting. As far as my observation goes, the excavation of a level disk proceeds for at least six months before it reaches the papillary border.

A true pressure-excavation, so affected that the lamina cribrosa is gradually pressed backwards, is always accompanied by an interruption of the nervous conduction. This is evidenced ophthalmoscopically by a discoloration of the excavation floor, and functionally by absolute scotomata. As is well known, the medial (inner) half of the visual field is first affected. Although I believe it possible that central defects may also result from pressure, I have had no experience of them. It is, however, important from a diagnostic standpoint to remember that every true pressure-excavation implies a defect of the visual field; conversely, even when the excavation encroaches upon the border of the disk and there is no scotoma, the pitting is not due to intraocular pressure.

In my opinion, the diagnostic value of the *halo glaucomatosus* is quite as small. It consists of an annular discoloration of the choroid surrounding the papilla, such as is quite often seen in normal eyes. I am convinced, as the result of an anatomical examination,[1] that the pale narrow ring forming the border of the glaucomatous pit results from a complete

[1] "Vorlesungen über den Augenspiegel," Berlin, 1864, S. 131.

atrophy of the choroidal circle that immediately surrounds the papilla. I found the choroid in this situation converted into a very thin, completely transparent membrane, such as may be seen in the advanced atrophy of axial myopia. There is, however, this difference: in extreme myopia the atrophic areas shade off into neighboring and less affected parts, while the glaucomatous ring stands out in sharp contrast with the normal choroid surrounding it. This atrophy in the case of glaucoma may be explained by the fact that sometimes the filaments forming part of the choroidal ring bury themselves in the lamina cribrosa, and when under the influence of increased tension the latter is pressed backwards the fibres are put upon the stretch, and atrophy results. The form of the atrophic area is seldom exactly annular, but is often irregularly indented towards the periphery, both by the encroachment of normal pigment as well as by the changes incident to axial myopia. It sometimes happens that the choroidal ring surrounding a glaucomatous excavation is deficient, so that the excavation appears as if surrounded by the sclerotic in the shape of a narrow, ring-like border. Nevertheless, the *halo glaucomatosus* may be absent about pressure-excavations, or it may be present unaccompanied by glaucoma.

Of those cases of optic-nerve excavation which are commonly called simple glaucoma, I have classified a fair number as distinctly glaucomatous. These are the cases that present a deep *unilateral* pit reaching to the margin of the disk. Inasmuch as the larger physiological cups are always bilateral, we may with certainty consider deep, one-sided excavations to be the result of increased intraocular pressure. Furthermore, into this class should be admitted only those cases that have normal iris movements—*i. e.*, distinct convergence-contraction, or, if the eye be blind, consensual contraction to light. If the iris in such cases of unilateral excavation be dilated and immovable, it is an indication of previous inflammatory attacks. This is true even when at the time of the examination no other traces of these attacks can be discovered, because outbreaks of glaucoma followed by paralysis of the sphincter iridis are always inflammatory

in character. This classification as glaucomatous of cases occurring without visible signs of inflammation (or at least of cases in which the diagnosis of a previous inflammation is merely inferred from present conditions), gives rise to a query whose answer must first of all be considered. The question relates to the development of glaucoma, and the conclusions can, of course, only be justified by the history which the patients themselves furnish. It is to be expected that the larger number have experienced the so-called initial symptoms, occasional halos, cloudiness of the visual field, etc., and many have had pains in the eye and over the forehead. The majority have at least had a variety of symptoms, while persons who are so innocent as to get blind in one eye without having meantime noticed anything remarkable, or who have discovered their blindness by accident or on being examined, can hardly be regarded as trustworthy in the presence of more reliable testimony. Finally, that these symptoms may be easily overlooked is proved by the fact that the halo-vision so common in iritis is rarely noticed by the patient. All patients affected by the so-called glaucoma simplex who had noticed their own condition have also remarked the occurrence of symptoms. *Whence I conclude that the course of the disease is marked by distinct attacks of increased tension that come on and (in the early stages at least) again subside.*

Whether these paroxysmal attacks are associated with inflammatory symptoms depends upon the particular case, just as in other ocular inflammations observation teaches us that one and the same pathological condition may be marked at one time by severe and at another time by very slight external signs of inflammation.

We may feel sure that the same intraocular conditions obtain in glaucoma as in irido-choroiditis, and that they vary in individual cases according to circumstances, and that the symptoms may be severe in some instances and barely noticeable in others. *The most important consideration is that the inflammatory symptoms of glaucoma are not the cause but the results of increased tension.* The rapidity with which it develops is more significant than the degree of intraocular

tension. Considerable increase of tension may be present without glaucomatous inflammation, but a sudden rise of intraocular pressure is a cause of inflammatory symptoms. As a proof of this, I quote the following case.[1]

K. G., æt. 66, was, January 5, 1865, admitted to the eye clinic of the Vienna University on account of acute glaucoma in her left eye, on which iridectomy was performed the same day. The patient thought her right eye was sound, but on examination tension was slightly raised. The a. c. was very shallow, and the retinal veins were markedly congested and tortuous. On the evening of the fifteenth day, after operation on the right eye, the patient was suddenly attacked by violent pains in the forehead and right temple, accompanied by a cloud of mist and showers of golden sparks before the right eye. Dr. Rydal found the eye free of ciliary injection, but remarkably hard. The pupil was dilated; media fairly clear; marked arterial pulsation; swelling and tortuosity of the veins; venous pulsation at the exit of the vessels; halo-vision; slight decrease of visual acuteness. There was an hourly increase of ciliary injection; œdematous swelling of lids and conjunctiva bulbi showed itself, and the media became more and more cloudy. Rest was obtained that night by a hypodermic injection of morphia. Next morning showed a completely developed attack of glaucoma; the cornea dull; the iris immovable; the pupil widely dilated; a high degree of tension; fundus examination impossible, owing to cloudy media. Iridectomy was immediately performed and recovery followed.

Cases of glaucoma that occur in the second and sound eye, while the healing process is still going on in the iridectomized eye, furnish us with an opportunity of confirming the foregoing observation. In Rydal's case it should be noticed that the increase of tension preceded the inflammatory symptoms, and was undoubtedly the cause of the latter. Instances in which the converse of this is true—where, although the intraocular pressure has disappeared, signs of inflammation persist—occur more frequently.

We may then conclude that so far as concerns those cases of glaucoma "simplex" that are really glaucomatous, but

[1] Dr. Lucian Rydal: "Ueber Glaucom."—Bericht über die Augenklinik der Wiener Universität, 1863-65. Wien, 1867. Bei Braumüller.

which at the time of the examination present no inflammatory signs, the careful observer will recognize in them the symptoms of the so-called "initial stage." The fact that nervous irritation, etc., may occasionally provoke glaucoma, proves that nervous influences may determine periodical attacks of increased tension. It is probable that in this lies the best explanation of the frequent occurrence, after an iridectomy for glaucoma, of the disease in the other eye. The ocular apparatus affords several examples of sympathetic secretion. A physiological instance is crying, a pathological one herpes frontalis. This limitation to sharply defined areas upon one side of the head is sufficient evidence that the outbreak of herpetic vesicles, as well as the inflammatory symptoms that accompany them, have a nervous origin. We know as little about their physiological connection as we do in the case of glaucoma.

The ciliary body is undoubtedly the locality in which the sympathetic secretion of glaucoma takes place. Here it is that liquids can most easily be poured into the vitreous body, and so increase its volume. It is possible that the first onset of glaucoma may be a very severe one and attended by disastrous results, but as a rule the disease is ushered in by a number of separate, mild, and transient attacks. The increase of intraocular pressure may, as observed in the so-called prodromal stage, be entirely transitory, but in the course of time it becomes a permanent symptom that may at all times be detected by comparison with the other eye. The picture of a glaucoma simplex with a slowly increasing intraocular tension, creeping on to blindness without variation in its signs and spmptoms, is a purely fanciful one. Of course no one can be prevented from believing in the existence of such cases, if he accepts as true a carelessly taken history, but the *proof* is still wanting.

The above-described slow and uninterrupted loss of vision in "glaucoma simplex" indicates, as might be expected, progressive optic-nerve atrophy with physiological cupping of the disk. The diagnosis is, of course, not always easy. Where one papilla shows a distinct excavation, while the

other disk is flat, the case is one of glaucoma without further question. But in the presence of physiological excavations the matter is often extremely difficult to decide. I have seen cases in which the pitting was large and bilateral, but not of the same size. While on the one side there was a narrow interspace between the pit margin and the papillary border, the other presented an exact picture of the so-called pressure-excavation, but vision was normal and the visual field uncontracted. I can only conclude from this that *physiological excavations are always bilateral, but are not always identical in size and location.* Such cases as these call for extended and careful observation. If the tension does not increase, if sight does not deteriorate, if the field of vision remains of normal extent, we may exclude glaucoma —it matters not how many "typical" signs of the disease may be present. With every excavation from pressure there of necessity go visual disturbances as soon as optic atrophy has developed. Although central vision may continue to be normal for a long time, restriction of the visual field finally shows itself, and this is equally true when the physiological excavation enlarges under intraocular pressure. Still more obscure is the diagnosis when a physiological excavation is complicated by an optic-nerve disease. Glaucomatous contractions at first always affect medial (inner) sections of the field of vision, but this may also occur in atrophic degenerations of the optic nerve. The color-sense may be unaffected in glaucoma, but this, too, is the case in some optic-nerve affections; the differences may be just as small as that between physiological and pressure-excavations. There are examples of undoubted glaucomatous excavations that do not extend to the margin of the disk, which it is not possible to distinguish from physiological cupping. In bilateral disease the determination of the tension is an uncertain diagnostic method. If one eye appears to be abnormally hard while the other is of normal tension glaucoma, is to be suspected, but + tension in both eyes proves nothing, inasmuch as normal tension presents the greatest individual variety.

The only road to success in diagnosis lies in continued

observation. First of all patients must be taught to note their own symptoms, especially whether they are subject to occasional attacks of cloudy or rainbow-like vision. If this is not practicable and excavation, accompanied by scotomata, is present, the question arises whether we should perform an iridectomy or not.

One cannot discuss the subject of iridectomy without thinking of von Graefe. How many thousands have been saved from impending blindness by his ingenious discovery! **As soon as the existence of glaucoma has been demonstrated iridectomy should be performed at the earliest opportunity.** One may temporize for a few days with eserine, etc., but, as a rule, the time so occupied is that much time lost. In some cases of glaucoma that come for treatment on account of inflammatory symptoms it is sufficient, as von Graefe[1] has mentioned, to excise a portion of the sphincter iridis. In proof of this I will cite the following case mentioned in von Graefe's article[2] on glaucoma:

Case 8.—Mrs. Maas, 60 years old, was treated by me in 1875. In February, 1857, she had been operated upon by von Graefe, first in the right eye, and five days later in the left. The left eye, which developed inflammatory symptoms shortly after the operation on the right, recovered with good vision. Eighteen years afterwards (April 5, 1875) the condition of this eye was as follows: the iridectomy, made inwards and somewhat downwards, is very small, barely 2 *mm* wide; the corneal incision is unusually long. Probably on account of the shallowness of the anterior chamber the operator was obliged to enter the keratome parallel to the plane of the iris, and so push it forward at an acute angle with the posterior surface of the cornea. The inner edge of the wound lies some distance from the scleral margin and is adherent to the iris throughout its whole extent. Vision is greatly reduced on account of a nuclear cataract, which was extracted July 30th. After discission of a secondary cataract, November, 1875, V with $+ 12$ D $= \frac{18}{50}$.

Although the patient lost her right eye some years later from optic-nerve atrophy, yet her visual acuteness continued

[1] *v. Graefe's Archiv*, Bd. xv., 3, S. 114.
[2] *L. c.*, Bd. iii., 2, S. 514.

to be good in spite of the anterior synechiæ, which must have prevented a restoration of the irido-scleral angle.

The operation is comparatively more effective in marked inflammatory cases than in those unaccompanied by signs of inflammation. In the former instance the visual disturbances are the result of several causes: (1) the corneal haze; (2) the pressure-paralysis of the retina; (3) when present, the optic-nerve excavation. The corneal opacity alone may reduce the vision to the counting of fingers held immediately in front of the eye. If, however, there be no restriction of the visual field, one may expect the patient will regain useful vision. In the absence of a cloudy cornea the reduction of visual power may be attributed to the optic-nerve excavation. Every excavation due to pressure is followed by contraction of the field of vision, although, of course, an optic-nerve affection with a physiological excavation is, as a rule, also accompanied by similar defects.

Von Graefe noticed[1] that the deterioration of vision following immediately after operation occurs in those cases of glaucoma whose scotoma stretches almost to the fixation point.

Astigmatism, due to the enlargement of the pupillary area (producing wider dispersion circles), as well as to the healing of the corneal wound, is to be held responsible for some interference with vision, and it is easy to understand how these effects are more detrimental in eccentric than in centric fixation. Even central vision is affected by both iridectomy and sclerotomy, yet this fact is not to be weighed against the lessened danger from blindness. These drawbacks are, of course, quite unconnected with the glaucoma; they have as important if not a much more important bearing on optic-nerve affections with physiological excavation of the disk.

I have quite frequently operated on cases of glaucoma having a very small field (about 10° radius) but with good central acuteness, and after the iridectomy there has been no further loss of visual power. It is otherwise with cases of optic-nerve atrophy with a normal excavation, as, of

[1] *v. Graefe's Archiv*, Bd. xv., 3, S. 182.

course, a progressive disease of the nerves is not to be checked by iridectomy.

In cases of glaucoma we may count with a probability approaching certainty upon a cure of the disease by iridectomy. Exceptions to this rule occur, but happily not very often. I have repeatedly seen cases presenting some glaucomatous symptoms even after a correctly performed operation.

CASE 9.—Mr. S., æt. 23, comes of a glaucomatous family. His father and uncle were operated upon by me for glaucoma. When first examined, February, 1883, patient reported

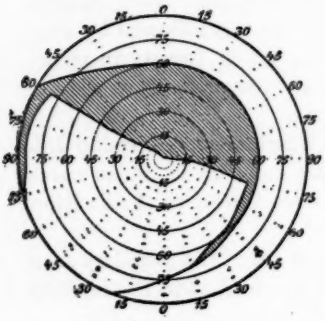

FIG. 8.

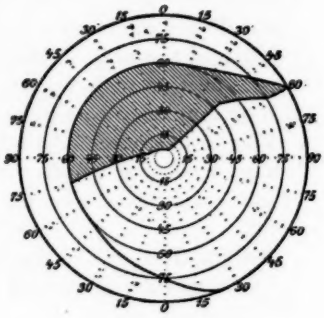

FIG. 9.

that he had had frequent attacks of rainbow-vision during the previous six months. V L = $\frac{5}{5}$; V R with — 0.75 = $\frac{5}{5}$; visual field not contracted; bilateral, deep, and abrupt excavations; angular bending of vessels at the papillary border. The halo-vision continued, and was unmistakably observed by the patient, who, as soon as he noticed anything wrong with his eye, was in the habit of striking a match to see whether or not there were any colored rings about the light. May 1, 1886, in both eyes V = $\frac{5}{5}$, but, in each, defects (see Figs. 8 and 9) in the upper half of the field, approaching the point of fixation. Cornea clear; pupil not dilated; the excavation (inverted image) reaches the upper margin of the nerve and thus corresponds completely with the situation of the scotoma; in the right eye there is (indirect examination) a *halo glaucomatosus* at the upper border of the nerve; the left eye in

this situation shows only a slight atrophy of the pigment epithelium. Bilateral upward iridectomy. In spite of this the rainbow-vision before the right eye continued, and in November gave rise to considerable anxiety on account of its frequency. In the floor of the excavation, also, distinct pulsation. Owing to the displacement of the blood-vessels only one—the artery—can be seen. November 15th, a second and downward iridectomy was done in the right eye. An unusual degree of astigmatism was the result, the keratoscopic image presenting vertically an elongated oval. The halo-vision persisted for some time but gradually disappeared.

But these cases do not always terminate so satisfactorily.

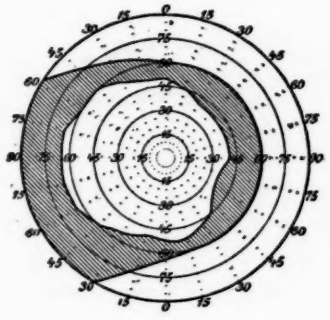

FIG. 10.

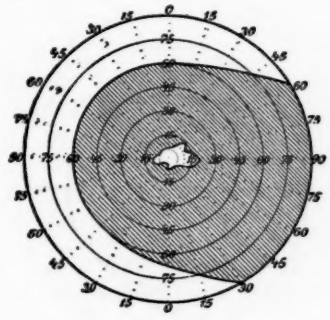

FIG. 11.

CASE 10. — W. T., æt. 46, treated October 20, 1886. Patient has had (right eye) cloudy, rainbow-vision for eighteen months. V R = $\frac{6}{18}$, + 0.75, 0.5 at 25 *cm.* Excavation reaching everywhere to border of disk; distinctly visible lamina cribrosa; sometimes spontaneous arterial pulsation. V L — 5 = $\frac{1}{12}$; 0.9 at 12 *cm.* Excavation to border of papilla. Broad peripheral iridectomy, both eyes. After this occasional arterial pulse and halo-vision. A year later the right eye became suddenly worse; pulsations in bottom of excavation; optic disk white; hand movements noticed only in a small portion of the field. April 16, 1890, spontaneous arterial pulsation, which again disappeared. Fig. 11 represents the right field of vision, taken before the iridectomy, October 20, 1886; Fig. 10, the left, April 17, 1890.

The following is a similar case:

CASE 11.—Mr. H. had for years cloudy and halo-vision. In December, 1873, V R = $\frac{18}{30}$; V L = $\frac{18}{30}$; defect in visual field towards inner side; bilateral excavation extending to margin. Patient could not be persuaded to submit to an operation until October, 1875. Hazy vision and rainbows continued; V, both eyes, was $\frac{18}{30}$, but the right-sided scotoma had greatly increased in size, and in addition to it, on the left side, was another up and in. October 23d, iridectomy. Patient still frequently complained of cloudy vision, particularly in the morning. The visual acuteness of the left eye was preserved, but in the right it was lost. Figs. 12 and 13 give the visual fields taken March 26, 1876. June 28, 1878, V R = $\frac{5}{30}$; V L = $\frac{5}{24}$. January 4, 1887, V R = fingers (above and out only) at about $\frac{1}{2}$ *m*; V L = $\frac{5}{18}$. April, 1891, the same.

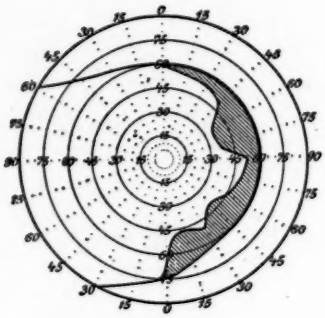

FIG. 12.

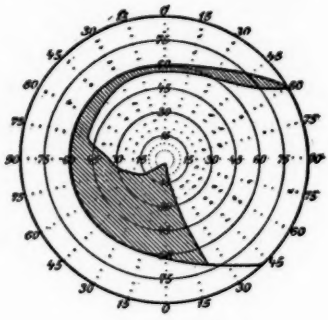

FIG. 13.

The statement that subsequent impairment of vision can be checked by repeated iridectomies or sclerotomies is not always justified by experience.

Although a malignant quality cannot be denied to those cases which do not exhibit the usual good results following iridectomy, the term *glaucoma malignum* is generally reserved for those that do not run a normal course after operation. On account of their rarity I consider it worth while to report those that have occurred in my private practice.

CASE 12.—Mrs. H., about 50 years old. Condition July 12, 1871: V L (M $\frac{1}{60}$) = $\frac{1}{4}$; + $\frac{1}{16}$ J No. 10. Scotoma in and below; good eccentric vision only, out and above; marked increase of

tension; media clear; pressure-excavation. R E $-\frac{1}{48}$; V $=\frac{1}{30}$; uncontracted field; optic nerve normal; no excavation, only a small natural pit. July 14th, on left eye a perfectly regular upward iridectomy. All went well until evening, then nocturnal pains, lachrymation, and injection of conjunctival sclera. Inflammation grew worse next day and developed a serious swelling of scleral conjunctiva, especially at inferior limbus, with a few conjunctival hemorrhages. On the pupillary margin of the discolored iris also a small hemorrhage. An empty anterior chamber and the eye harder than before the operation. After about four weeks the inflammatory symptoms subsided and the tension had diminished somewhat, but the a. c. continued empty, and during the later course of the disease the left was subjected to frequent inflammatory attacks. Last examination end of Nov., 1872; the a. c. in same condition; cornea irregular through loss of epithelium. Lens reflex very marked; ophthalmoscopic examination showed centre of pupillary area hazy, only the periphery transparent; fundus not visible. Anterior perforating veins enlarged. The eye is still hard. No cystoid cicatrix, only a small transparent vesicle in the scar. Right, condition unchanged.

Case 13.—Mrs. O., æt. 60, a depressed, nervous individual. May, 1872, V R $-\frac{1}{36}=\frac{1}{18}$; scotoma up and in; commencing excavation; T +; frontal pains; halo-vision; V L $-\frac{1}{48}=\frac{1}{18}$; normal field and no excavation. Oct. 28th, excavation almost complete. Nov. 13th, V $=\frac{1}{18}$; $+\frac{1}{18}$ J No. 5. Nov. 18th, puncture of a. c., which immediately filled. Nov. 21st, upward iridectomy, followed next day by pains, and later on by swelling of upper lid and symptoms as in foregoing case. Wound did not close until Dec. 15th. The a. c. still remained empty, although the media became clear. Vision reduced to perception of light in a small, external portion of field. Tension normal. Five years later: most of the right iris attached to the cornea; anterior chamber very shallow or empty; V L (M 1.5) $=\frac{5}{6}$; no excavation.

It is worthy of note in this case that although the paracentesis wound healed smoothly, the iridectomy resulted so disastrously.

Case 14.—Miss M., æt. 48, advanced case of glaucoma in left; iridectomy followed by severe inflammatory reaction; cystoid vesicles in scar and total loss of vision.

CASE 15.—Mrs. L., æt. 65. July 2, 1872: right eye glaucoma absolutum. Thirteen years before von Graefe did an iridectomy on this eye, after which, according to patient's story, healing was slow and irregular. Left eye growing worse since Christmas, 1871. Glaucomatous symptoms not well marked; V = $\frac{1}{40}$. Iridectomy, July 12th, in good form. In eight days posterior synechiæ; later, hemorrhage in iris at pupillary border. Vision grew worse until, in the middle of August, it was reduced to finger-counting at five feet. Lens reflex; empty a. c.; eye hard. Last examination, Jan. 23, 1873: cystoid cicatrix; a. c. almost empty; nuclear cataract but equator lentis clear in iridectomy region; V = fingers at 1–2 feet.

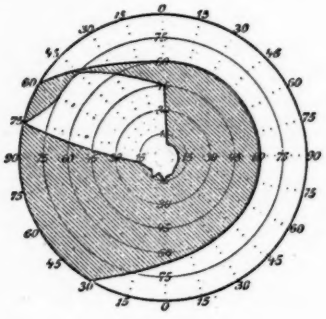

FIG. 14.

CASE 16.—Miss F., aged 47. V L = 1. Right: commencement of blindness not noticed; temporary cloudiness; pains; no halos. Jan. 13, 1875, V = fingers uncertain and eccentrically; large scotoma; deep excavation; pupil not dilated and media clear; marked increase of tension. Jan. 19th, iridectomy. Eye still hard; much lachrymation and conjunctival hyperæmia. The a. c. remained empty. March 5th, vision reduced to bare perception of light. April 23, 1878, iris applied to cornea; eye atrophic; cataract. Left, still normal.

CASE 17.—Mrs. W., aged 49. Aug. 3, 1876. Three years before, "burning" in right eye; gradual loss of sight; excavation with white background reaches to edge of disk, + T. *Left*, glaucoma with marked inflammatory symptoms. V = $\frac{1}{4}$; F of V restricted as in Fig. 14. Aug. 4th, iridectomy, both eyes. Iris angles well replaced. In spite of careful treatment and watching,

both eyes went from bad to worse until Jan., 1877, when V L = hand movements; a. c. empty; cloudy cornea. March 19th, left, same vision; right, no perception of light.

CASE 18.—Mrs. B., æt. 45. June 28, 1881: left, for one half year, halos and cloudy vision; V = $\frac{1}{3}-\frac{5}{12}$; large scotoma and excavation; cornea clear; no dilatation of pupil; + T. Right: V = $\frac{5}{6}$; optic nerve and F of V normal. July 1st, iridectomy; left, small anterior synechia, but angles free. Sept. 17th, V L = fingers at 1 *m*; scar broad and dark-colored; a. c. shallow. March 6, 1882, left, complicated cataract; right, normal.

One case which I was at first disposed to regard as an example of glaucoma malignum I now consider to be of the same nature as those cases of irido-choroiditis terminating in blindness that in pre-antiseptic times (to which it as a matter of fact belonged) so frequently followed cataract operations.

The most tedious restoration of the anterior chamber is not necessarily evidence of glaucoma malignum since it is commonly encountered in cases where increased tension has persisted for a long period. The aqueous chamber may remain empty for six to eight days without evil consequences. Nor is its evacuation after iridectomy in glaucoma malignum the cause of the subsequent trouble. In Case 13, for example, where a paracentesis corneæ was done several days before the operation, the chamber immediately refilled, although it remained a long time empty after the subsequent iridectomy.

In addition to the foregoing cases of glaucoma malignum I have seen others not operated upon by me. From this varied experience I deduce the following rule: *When glaucoma malignum attacks one eye, it follows iridectomy on the other eye, even when the second eye is not affected for years after the first.* The cause of the unsatisfactory results of the operation must lie in some obscure structural anomaly inherent in the eye itself. *Experience teaches us that in all cases of chronic glaucoma affecting both eyes it is advisable to operate first of all upon the worse one, even if it be absolutely blind. Should this be followed by the normal-healing process the second eye may be operated upon without the least apprehension.*

Fortunately, cases of glaucoma malignum occur so rarely that special mention of them is made in glaucoma statistics. All my private cases presented themselves between 1871 and 1881, and up to the present time I have not had another.

In any event these rare cases of glaucoma malignum furnish no excuse for preferring sclerotomy to iridectomy. *We may count upon a favorable result following iridectomy with almost perfect certainty, while sclerotomy, on the other hand, is by no means an infallible remedy.* In support of this opinion I can furnish a number of examples.

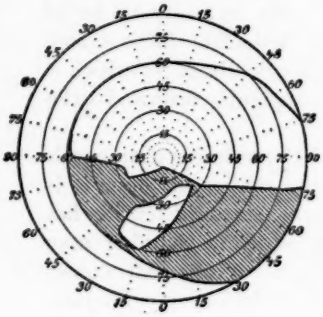

FIG. 15.

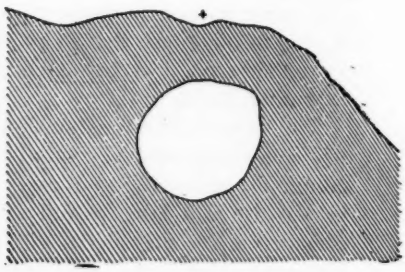

FIG. 16.

CASE 19.—I. S., æt. 33, March 11, 1878, *left*, rainbows for six months; cornea clear; small pupil; V = $\frac{6}{9}$; uncontracted field. Right, V = $\frac{6}{12}$. Large physiological excavation of both disks. July 1, 1880, *left*, V = $\frac{6}{24}$, 0.6 slowly at 20 *cm*; + T; peculiar inferior scotoma that encloses an unaffected retinal area. This is represented in Figs. 15 and 16, the first a perimetric measurement, corresponding to a control projection (Fig. 16) on a plane surface. Halo-vision more frequent; cornea clear; pupil not dilated; R V = $\frac{6}{12}$, 0.24 at 20 *cm*; normal field; peripheral vision good. July 30, 1880; left, sclerotomy. May 24, 1881, left, V = peripheral (best outwards), finger counting. Right, V = $\frac{6}{18} - \frac{6}{12}$; peripheral V good.

Sclerotomy did not influence the course of the glaucoma. Of course the subsequent iridectomy performed on the left eye did not effect an improvement.

CASE 20.—Mrs. St., æt. 64, July 18, 1876. A year ago a sudden cloud before the right eye, lasting several months. No reliable history; clear cornea; pupil not dilated and consensual movements normal, but no reaction to right; deep excavation; + 4 D, No. iv. Sn. at 10″. Small central F of V, contracted as per Fig. 18; + T. *Left:* V + 4 = $\frac{5}{15}$–$\frac{5}{6}$; no excavation and *F of V unrestricted.* July 27, 1876, right, iridectomy. September 22, 1876, right + 6 = letters of Sn. vii. Left, + 4, V = $\frac{5}{15}$; + T. Slightly hazy vision; some pain in ocular region; no excavation. March 12, 1877: R + 6 = No. 4, 30 *cm*; V = $\frac{3}{40}$, L + 4 = $\frac{5}{6}$. February 18, 1881: Slight glaucomatous attack; *left, sclerotomy*, February 21st. March 10th: L + 4 = 0.4 at 15 *cm*. March 14th: cloudy vision and pain. April 17th: L V + 4 = $\frac{5}{15}$; R V = doubtful perception of light. July 15, 1882: left + 6 = 0.3 in 20 *cm*; beginning excavation.

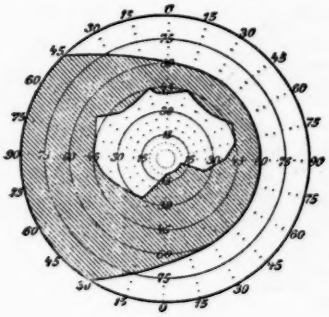

FIG. 17.

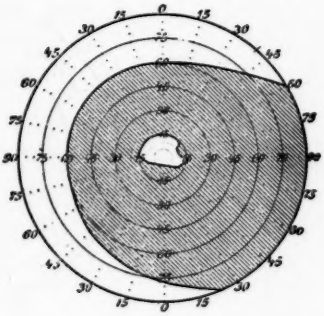

FIG. 18.

The left eye grew progressively worse until finally on December 24, 1883, an iridectomy was done. The visual field at that time was as shown in Fig. 17. May 24, 1885, V = $\frac{5}{36}$; beginning cataract.

The sclerotomy did not check the progress of the glaucoma, which, however, was accomplished two years later by an iridectomy.

CASE 21, Mrs. K., aged 54. July 21, 1883: R V + 2 = $\frac{5}{6}$; scotoma above and in. L V = hand-movements up and out only; *right*, sclerotomy; *left*, iridectomy. August 24th: *Right*, sharp attack of cloudy vision. March 31st: V R + 2.5 = $\frac{5}{15}$; large scotoma as in Fig. 19.

April 3d: right, iridectomy. June 21, 1886: V R + 2 = $\frac{5}{6}$.

In this case also the patient was not relieved by sclerotomy. For a year after that operation the attacks of glaucoma persisted. The field of vision became more and more restricted, until finally the diseased process was brought to a standstill by an iridectomy.

I have not observed attacks of glaucoma occur after iridectomy, as I have seen them set in after sclerotomy has been performed. Apart from glaucoma malignum, cases of blindness from glaucoma in which an iridectomy had been done, are extremely rare. In my experience, examples of the inefficiency of sclerotomy in this disease are quite common. Observation has convinced me that it is not to be

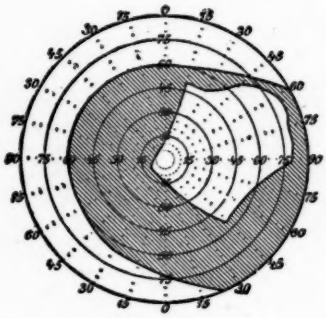

FIG. 19.

depended upon in cases of true glaucoma. It has been employed most commonly in cases of so-called glaucoma simplex; and it is possible that many a case of stationary or slowly progressive optic-nerve disease with a physiological excavation has been put forward as proof of its curative value. Moreover, the length of time these reported cases of cure had been under observation is much too short. One or two years is not a sufficiently long period when we know that many cases of glaucoma are of much longer duration; the following case, for example:

CASE 22.—Mrs. J. first presented herself for treatment in 1874. For four or five years previously she had suffered (mostly in the evening) from cloudy vision affecting her left eye. After a

night's rest the attack disappeared. The first severe symptoms were noticed February 20, 1874. These were ciliary injection, mydriasis, corneal haze, etc. V = fingers at 1½ *m*. Iridectomy, February 25th. A few days later the right eye was also seized. Patient refused iridectomy; attack soon over. March 24, 1874: L V + 4 = $\frac{10}{30} - \frac{10}{20}$; R V + 5 = $\frac{10}{10} - \frac{10}{15}$. Repeated glaucomatous attacks in *right*, especially a marked seizure in January, 1883. V + 5 = $\frac{1}{3}$. Eserine induced complete myosis. V + 5 = $\frac{1}{2}$. On February 6th another attack; then iridectomy and relief. February 8, 1888: V R and L + 5 = $\frac{6}{18} - \frac{6}{20}$.

In the right eye the so-called "initial stage" had lasted about nine years, from February, 1874, to February, 1883, the date of the iridectomy. Had a sclerotomy been done at the time of the first attack, the operation might have been given the credit of relieving the patient for that length of time.

The following history shows the untrustworthy character of sclerotomy.

Case 23.—Mrs. W., aged forty-five, came December, 1882, on account of cloudy and rainbow-vision. V R = $\frac{5}{6} - \frac{5}{6}$; V L = $\frac{5}{6} - \frac{5}{6}$. The symptoms were regarded as evidence of glaucomatous attacks, and on December 15th *sclerotomy* was done in both eyes, but the halo-vision and cloudy sight still persisted, until January, 1885, a severe attack of glaucoma affected the left eye, so that V = finger-counting at ½ *m*. After a bilateral iridectomy no more evidence of the disease. April, 1887, V L = $\frac{5}{6}$.

Finally the same disagreeable results may follow both sclerotomy and iridectomy, at least I have seen after sclerotomy retinal hemorrhages so profuse that it would have greatly interfered with any remnant of visual acuteness.

From all this we can only conclude that the essential part of glaucoma operations is the excision of a portion of the iris. It is true that we are ignorant of the real connection between the two, but it is not the province of physical science even to discuss stories that will explain all the phenomena. He who reasons like a *physician* is satisfied therewith. When the array of facts is complete, their interpretation follows as a matter of course.

If the iris is not attached to the cornea, but simply pushed forward as a consequence of increased volume of the vitreous, we may certainly regard the former condition not as a cause, but as a result, of the glaucoma. One may frequently observe in the course of the disease that attacks disappear spontaneously, even while the iris is bulging. The iris resumes its normal position without affecting the further progress of the glaucoma.

Among other untenable propositions which Leber advanced at the International Ophthalmological Congress (Report, 1888, p. 278), the following may be mentioned: "When the filtration angle becomes entirely occluded, no operative procedure is of any value—as is the case in the later stages of glaucoma." This assertion has no foundation in fact, because no man can say whether a complete stoppage of the irido-scleral angle is present during life or not. Experience teaches us not to count upon any good results from an iridectomy if the conducting power of the optic-nerve fibres be destroyed, but that has nothing to do with the filtration angle.

The only definite physiological fact of any value relating to the action of iridectomy in glaucoma that has yet been made is the observation of Exner,[1] that at the border of the coloboma a direct anastomosis between the arteries and veins of the iris is rendered possible, "so that the arterial blood without passing through an intervening capillary area may at once reach the veins." The blood pressure in the iris must in this way be at least moderated: but the claim that this alone suffices to cut short the glaucomatous process is, in my opinion, rather premature.

As soon as an iridectomy has been performed on a genuine case of glaucoma, we may regard it as certain that the disease has been brought to a standstill. The instances in which this is not true are so extremely rare that from a statistical standpoint they do not carry much weight.

Though iridectomy is a safeguard against glaucoma, there are doubtless cases in which marked inflammatory attacks of the disease affect iridectomized eyes. I can recall but one

[1] *Wiener medic. Jahrbücher*, 1873.

instance in my own experience. I have already reported this case,[1] but will again refer to it at some length.

CASE 24.—Mrs. A., æt. 66. First saw her July, 1878. Left eye: (H 5.5) V = $\frac{5}{6}$. Right, mature cataract. November, 1882, right: keratitis, œdematous infiltration and iritis, with numerous synechiæ. After four weeks' treatment with atropine the inflammatory symptoms disappeared, leaving several posterior synechiæ—two small ones above, a broad one below. In July, 1885, extraction of the cataract in right eye with a broad iridectomy. The coloboma was perfect, and there was no incarceration of the iris; the inferior synechiæ of course remained. February 8, 1886, the patient returned, on account of pains which she had for several weeks in the left eye. There was a small amount of ciliary injection; some infiltration and haziness of cornea; + 12, V $\frac{1}{12}$ – $\frac{1}{18}$, corresponding to the amount of corneal cloudiness; atropine. The patient lived outside of Berlin, and did not again return until March 6th, with condition but slightly changed. In place of the cornea infiltration there was some loss of substance. At the next examination, March 16th, the area of corneal haze was small, its lower segment showing a superficial loss of substance. Fundus examination, now possible for the first time, revealed marked arterial pulsation, as well as a deep excavation extending to the disk border. Owing to contractions of the orbicularis it was not possible to determine the ocular tension, but after the use of cocaine it was found to be distinctly increased. Left eye: Tn. Hand movements perceived out and above only. In addition to the keratitis superficialis, etc., the patient had glaucoma, which would certainly have been diagnosed earlier had she presented herself with greater frequency. Later, extraction was performed on the left eye. Then discission of a secondary cataract, a prolonged convalescence due to inflammatory complications, and finally a good result.

I lay special stress upon the fact that extraction of the right lens was followed by normal convalescence, and that the eye was not affected by glaucoma until eight months afterwards. Schmidt-Rimpler[2] has observed a similar case. Three months after a perfectly successful peripheral linear

[1] These ARCHIVES, vol. xvii., 2, p. 140.

[2] Graefe-Saemisch: "Handbuch der gesammten Augenheilkunde," Bd. v., S. 49.

extraction (rather large coloboma, no iris incarceration) the eye was attacked by acute glaucoma. There were no initial symptoms. Even in this case the iridectomy was of value. It is worthy of note that as a result of previous inflammatory attacks there were extensive anterior and posterior synechiæ.

There are several cases reported in literature in which glaucoma is said to have affected aphakic and iridectomized eyes before the extraction wound had completely healed. I cannot help suspecting that these symptoms, which appeared during convalescence after extraction, were not as closely observed as they might have been. To this category belong many cases which Treacher Collins[1] has well designated "cases which for want of a better name may be termed glaucoma after extraction."

This term may well be applied to certain cases of intraocular inflammation which are conveniently termed glaucoma for want of a better word. But it is not to be wondered at when eyes (whose ailments are styled glaucoma simply because it has an attractive sound) are enucleated to afford opportunities for anatomical research, that they furnish little or no information.

Hydrophthalmus is also a form of glaucoma, and has been recognized as such since Horner demonstrated that it may lead to excavation of the papilla and blindness. During the first stages of hydrophthalmus in early infancy I have noticed the repeated advances and retreats of glaucomatous attacks, the increased tension, the ciliary injection, and the hazy cornea, and recognize in hydrophthalmus a result of glaucoma occurring in infancy and childhood. The increase in the volume of the vitreous stretches the non-resistent sclerotic of infancy. That there is such a thing as a congenital hydrophthalmus is undoubted, and it is common enough to call every case of hydrophthalmus in children congenital. The youngest child that I have ever seen affected by the disease in one eye only was three months old. Whether it was congenital or not cannot of course be decided. This affection must not be confounded with the still rarer *cornea globosa*—

[1] Ophthal. Hospital Reports, 1888, p. 19.

that is, an abnormally large cornea with a deep anterior chamber, but without stretching and thinning of the sclera.

I have repeatedly performed iridectomy in children affected by hydrophthalmus, and have not found the dangers said to attach to such a procedure at all realized. One should, however, avoid the mistake of making a too peripheral incision, which ought to lie along the corneal border of the limbus. In illustration, I report a few cases.

CASE 25.—R. S., 7 months old, was brought to me March, 1887, with bilateral hydrophthalmus. Iridectomy was finally done on the worst (right) eye the end of December. Four weeks later the left eye presented a dense corneal haze, deep a. c., and sensibly increased tension. For this, an iridectomy was done in January, 1888. Recovery absolutely normal in both eyes. In February, 1889, both corneæ were clear, and visual acuteness, as far as could be determined in a child barely two years old, was good.

It can be proved that hydrophthalmus frequently begins in childhood.

CASE 26.—F. W., æt. 7, came in May, 1889, with hydrophthalmus of the right eye. The disease had been discovered two years before. The sclera about the cornea is stretched, the cornea itself is enlarged and presents a number of opacities; the pupil is wider than its fellow; deep excavation of the nerve; V = $\frac{1}{24} - \frac{1}{18}$. Left, normal; no excavation May 15th; iridectomy *right*, with normal recovery.

As in glaucoma, so with hydrophthalmus, the only remedy that will prevent blindness is an iridectomy made as early as possible. The technique in the latter case is somewhat more difficult than in glaucoma, and it is not easy to draw conclusions as to the final results, since the tender age of the patient precludes a satisfactory test of vision. In the cases operated on by me, convalescence was with few exceptions rapid and normal. Intraocular hemorrhage after iridectomy occurs very rarely.

I will not say more about *glaucoma secundarium* than that it represents too motley a crowd; often the term so used is devoid of any definite meaning. But two groups are distinguishable, the so-called hemorrhagic glaucoma, and glau-

coma due to occlusion of the pupil and bulging of the iris periphery.

That glaucoma follows retinal hemorrhage (rarely indeed, though often enough to establish some sort of connection between them) has long been known, but neither the bleeding nor the glaucoma has anything characteristic about it. We know as little why retinal hemorrhage is or is not followed by glaucoma, as why or when (especially if a cloudy cornea prevents an ophthalmoscopic examination) the disease exhibits the hemorrhagic complication. As in primary glaucoma, the hemorrhagic form occurs most frequently in hypermetropia, more rarely in emmetropia, and least often in myopia.

According to reports of many cases, the glaucomatous outbreak occurs a short time—a few days—after the retinal hemorrhages. It may be said in this connection that one cannot tell how long they may have been present. It may easily happen that peripheral bleedings have occurred a long time previously, in which case the patient would not notice them until a hemorrhage in the macular region led to an ophthalmoscopic examination. As a rule, the glaucoma follows the hemorrhage after one to two months, the longest interval, so far as my observation goes, being eight months.

Case 27.—Mr. S., æt. 72, was treated October 2, 1876, about eight days after he had noticed certain peculiar "glittering spots" before his right eye. Right eye: No. 5.5 is slowly read. Numerous small (diameter $\frac{1}{3}$ of the disk) retinal hemorrhages, some in macular region. Retinal veins tortuous; retina unclouded; vitreous clear; V L (M $\frac{1}{18}$) = $\frac{6}{12}$; fundus normal. May 29th: sight much worse than before, the retina about the optic nerve hazy, and the vessels, especially the arteries, very small. More hemorrhages, some of which appear quite recent. June 5, 1877: Pain and marked pericorneal injection for past eight days; cornea cloudy; iris unchanged; atropine.

June 10th.—Atropine mydriasis; moderate pains; considerable injection of scleral conjunctiva; cornea very hazy; no view of fundus; marked increase of tension. Eserine 1 per cent. June 16th: pupil somewhat irregular but of normal size; otherwise the situation unchanged. Finally, July, 1877, the continuous

pain affected the patient's health and the lost eye was enucleated. The result of the anatomical examination is given by Deutschmann.[1]

It goes without saying that the prognosis in hemorrhagic glaucoma is more unfavorable than in retinal hemorrhage. The injury to sight due to the hemorrhage is brought about by a tearing apart of the nervous elements within the substance of the retina, and of course is irreparable; but our experience of hemorrhage setting in after iridectomy teaches us that the retention of good vision is compatible with retinal bleedings. In cases of glaucoma hemorrhagicum the most important question relates to the amount of damage done to vision by the retinal hemorrhage before the glaucoma set in, because the most that can be hoped for is the restoration of the amount that was then present; if there has been useful vision it is our duty to attempt to preserve it. *Iridectomy in glaucoma hemorrhagicum has acquired an undeservedly bad reputation.* Although in some cases under my care a well-timed operation had preserved the visual acuteness, there were others where iridectomy did no good. These latter were instances in which sight had already been lost. I will report some of them.

Case 28.—Mrs. K., aged 64. September 11, 1873 R H $\frac{1}{20}$; V = $\frac{1}{30}$, + $\frac{1}{10}$ Sn. No. 2 : 12″ L : four weeks before, the house physician noticed that the left eye had become blind from retinal hemorrhage. In addition to this she became the subject of a painful inflammation of the eye, the pains being situated in and over the eye. The cornea is cloudy, the pupil (after atropine) somewhat dilated and fixed by posterior synechiæ. The scleral conjunctiva is injected, but the anterior perforating veins are not dilated. Slight increase of tension. Fingers counted eccentrically in all directions. Fundus not visible.

As the pain increased in violence during the next day, I advised enucleation, but the patient would consent to an iridectomy only. Accordingly, on September 30th, a broad, regular, downward iridectomy was done under chloroform, with the keratome. Shortly after the bandage had been applied and the patient had recovered consciousness, vitreous presented under the dressing.

[1] *v. Graefe's Archiv*, Bd. xxv., 3, S. 163.

This was probably the result of straining (from vomiting). Severe pains now set in, which were relieved by applications of ice. No doubt an intraocular bleeding had occurred, although this much-dreaded complication is extremely rare. Swelling of lids and conjunctiva followed, with moderate pain. These soon disappeared after treatment with lukewarm fomentations. October 5th: there is no more swelling of the lids; some blood in the anterior chamber. Later on, bulging of the cicatrix, cataract, and complete adhesion of the pupillary border; the a. c. remained empty. The pains are slight; some persistent increase of tension.

In July, 1874, these symptoms were for a time more urgent, the inflammatory condition more marked, and one spot, in the upper and inner ciliary region, painful on pressure. Finally atrophy set in with flattening of the globe in the direction of the recti muscles. No more complaints.

The condition of the right eye entirely unchanged.

Last examination, March 24, 1879: Left, phthisis bulbi; ciliary soreness above, occasionally also spontaneous pains.

R + $\frac{1}{12}$ No. 0.5 – 0.75. Papilla not excavated; F of V uncontracted; commencing nuclear cataract.

In another and more disastrous case of this kind, where the author did an iridectomy in one eye and Hirschberg operated on the other, malignant glaucoma set in and complete loss of sight was the result. In this instance there was a complication of retinal hemorrhage with malignant glaucoma, from which one may conclude that one affection does not necessarily exclude the other.

I have notes of six cases in which the iridectomy wound healed readily and without complication, but either more acuteness of vision or less resulted than might have been expected. One of them I give.

Case 29.—Mrs. M., aged 47, came March, 1887. V L (H 1.5) = $\frac{6}{12}$. Oph. normal. R: for three weeks vision getting worse; numerous retinal hemorrhages. V = fingers at 15 *cm.* Middle of May: R: pains in and redness of the eye. 26th: smoky haze of cornea; pupil dilated and fixed; marked increase of tension; fingers counted at 10 *cm;* hand movements perceived eccentrically everywhere.

June 19th.—Iridectomy followed by normal recovery.

In all these six cases the purpose of the iridectomy was completely accomplished, *i. e.*, the pains were relieved and whatever remained of sight was preserved. When useful vision is still present in a case of glaucoma hemorrhagicum, it can be maintained by means of iridectomy.

On account of the presence of a pressure-cavity it is legitimate to speak of secondary glaucoma as following iritis. The conditions present are complete occlusion of the pupil and bulging of the iris periphery.

Glaucoma and iritis are, of course, not mutually exclusive, and they may develop together, as in cases that have come under my observation.

I know of others, also, that had been treated by atropine on account of synechiæ until they became blind from glaucoma. The corneal cloudiness in such cases is quite the same as that occurring in glaucoma; the atropine mydriasis excites no comment, the fundus is not visible, and the examination of the tension is really the only means available for a differential diagnosis.

Quite frequently, on the other hand, one sees loss of vision occur in cases of pressure-excavation where complete occlusion of the pupil and pushing forward of the periphery of the iris had developed. It is when iritis is followed by these additional symptoms that glaucoma is especially to be dreaded. Its cause lies in the dragging upon the ciliary body. If, however, this state of affairs comes about slowly and without noticeable inflammatory signs, there is, in the early stage at least, no danger of glaucoma, especially when a prophylactic iridectomy is made as soon as possible. To show how pupillary occlusion with prominence of the iris periphery can develop without leading to glaucoma, I report the following case:

Case 30.—Mr. G., about 30 years old, consulted me, middle of May, 1882, about a right-sided iritis which had already lasted three weeks. The patient was treated with atropine locally, and, on account of fibrinous exudation in the a. c., was ordered Turkish baths with good results. About June 15th a return of the iritis. The end of July the disease had disappeared; V — 0.75 = $\frac{6}{6}$. June, 1887, another iritic attack with a small posterior

synechia in the upper part of the pupil. End of July, 1890, still another attack of iritis in the right eye. This time there was marked conjunctival hyperæmia with some corneal haze and pain. Atropine and duboisine produced complete mydriasis except in the region of the synechia above mentioned; V — 0.75 = $\frac{5}{25}$; increased cloudiness of cornea; lessened mydriasis. Finally, after a severe and prolonged attack, although the mydriatics were discontinued, the pupil remained dilated. The V gradually recovered and was from $\frac{1}{18}$ to $\frac{5}{15}-\frac{5}{10}$ the middle of October. Towards the end of November, on account of some accommodative paresis, eserine was instilled, but was stopped in December, having meantime produced a marked but temporary contraction of the pupil. Jan. 10, 1891, V R — 1.5 = $\frac{5}{15}-\frac{5}{10}$. Pupil in same condition as before, but is funnel-shaped, its entire border being bound down to the anterior capsule. Its periphery is everywhere bulging, but there is no conjunctival hyperæmia, no increased tension, no pain, etc. January 14th, iridectomy. January 28th, V R — 1 = $\frac{5}{15}$. This iris is now in its proper plane, and on careful examination the site of the pupil is seen to be occupied by a delicate membrane dotted over with fine dark points. The artificial pupil is black and clear.

The bulging of the iris periphery had in this case lasted a full week without the faintest sign of glaucoma.

The claim that blocking of the filtration angle in the anterior chamber is the cause of glaucoma is put forward with all the authority of a physiological law. But natural laws permit of no exceptions, and it must necessarily follow, if Leber's theory were correct, that every case of a narrowed irido-scleral angle is followed by glaucoma. But this is by no means true. Every anterior synechia, inasmuch as it brings about a lessened filtration angle, would result in glaucoma, yet the fact is that these two conditions are almost never associated.

Priestley Smith[1] puts forward in support of the filtration-angle theory the following clinical evidence: "A soft, transparent lens, incompletely attached at the zonula, slips into the anterior chamber and lies in front of the pupil. Inside of an hour a severe glaucomatous seizure. Next day the lens returns to its place and the glaucoma disappears. This

[1] International Congress at Heidelberg, 1888, p. 231.

is repeated upon several occasions and is followed as often by a return and a subsidence of the glaucoma. Finally the lens is extracted and the glaucoma does not return." Dislocation of the lens into the anterior chamber is a well-known cause of severe and painful inflammatory symptoms which I would prefer to regard as an incipient irido-choroiditis.

Glaucoma presents an entirely different picture. That an anteriorly dislocated lens is capable of completely obliterating the filtration angle is at least a rather strong statement. In my opinion the backward pressure of the lens *against* the iris *from* the cornea would rather tend to enlarge it. Moreover, the lenticular diameters are less than those of the anterior chamber. If the body of the lens, only partially

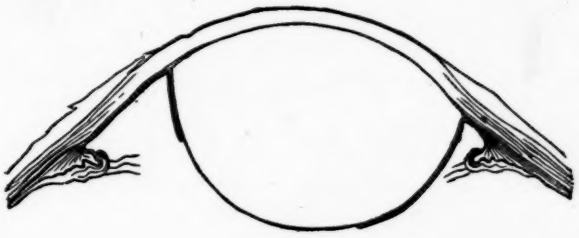

FIG. 20.

occupying the anterior space, produces glaucoma, why does not every anterior synechia bring about a similar result? Fig. 20 (*l. c.*, p. 232) represents the diagram with which Priestley Smith illustrated his case, but, as Samelsohn (*l. c.*, p. 280) observed, during the discussion that followed, it is not a faithful picture of recent cases of the kind. Adhesion of the iris to the cornea, as shown in the picture, may occur later, but it is a consequence of the inflammation and not a cause of it.

Dislocation of the lens *into the vitreous* is said to produce secondary glaucoma. I consider many of these cases to be traumatic in character, while others are members of that group that has been labelled glaucoma "for want of a better name."

For the purpose of making the filtration-angle theory do service in the case of vitreous dislocations of the lens Priestley Smith (*l. c.*, p. 282) pictures the condition repre-

sented by Fig. 21. The filtration angle is said to be obliterated on one side by pressure of the lens on the ciliary body, on the other side by the displaced vitreous. One may well ask, what makes the lens press on the ciliary body? The weight of the displaced lens is all that can be exerted upon it, and that the ciliary region can stand. If the normal pressure of the vitreous body leads to glaucoma, every man and every other animal would have the disease.

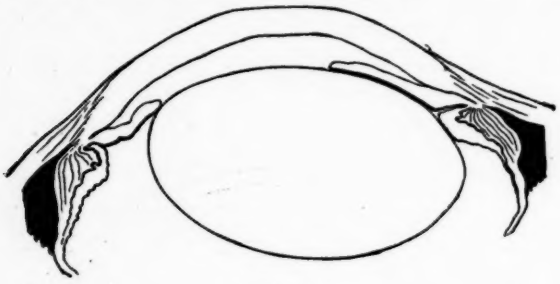

FIG. 21.

That lenticular luxation may exist for years without evil consequences is shown by the following case:

CASE 31.—Mr. L., æt. 44. September, 1874, V R = $\frac{10}{20} - \frac{10}{40}$; V L = 1. In May, 1875, the right lens was cloudy throughout. In June, 1875, it was noticed to be movable and dislocated downwards. Beginning of August, with + 10 D V = $\frac{1}{30} - \frac{1}{40}$. In October it was completely displaced downwards and visible as a light-gray body. The vitreous is normal; only a slight circumscribed haze near the papilla. July, 1880, V R + 10 = $\frac{5}{12} - \frac{5}{8}$; left, irregular striæ in anterior cortex; V = $\frac{5}{8}$. January, 1883, V R + 10 = $\frac{5}{8} - \frac{5}{8}$; left, mature cataract. The left lens gradually sank into the vitreous. January, 1890, V L + 9 = $\frac{5}{18}$.

This patient, according to Priestley Smith, should have had glaucoma, but he did not. The condition just referred to, long recognized and first described by Kniess,[1] is no proof of the assertion that it is a cause of glaucoma; there is abundant evidence to prove that it may exist without glaucomatous symptoms. Clinical evidence, for example: the anterior chamber is sometimes so shallow in interstitial

[1] *v. Graefe's Archiv*, Bd. xxii., 3, p. 163.

keratitis that anterior synechiæ may form and the filtration angle be obliterated; yet no glaucomatous signs develop. On the contrary, the tension is usually minus in such cases. Likewise, Leber's theory, that in eyes enucleated because of glaucoma the drainage from the anterior chamber is considerably lessened or entirely choked proves nothing, except that an enucleated glaucomatous eye remains harder a longer time than one not affected by glaucoma, because the filtration angle is obliterated by the glaucoma, in consequence of the increased volume of the vitreous.

The experiments by Leber[1] on the filtration of fluids at the irido-scleral angle are post-mortem experiments on animals, and of little value when living eyes are under consideration. His experiments on the latter simply show that a *slight* increase of intraocular pressure may disappear, but marked tension not. It does not follow from this that gradual decrease of tension with a moderate increase of pressure comes about in consequence of filtration through the filtration angle, for if the manometric pressure in the anterior chamber is increased absorption takes place, as Donders'[2] experiments show, as a result of the communication of the pressure to the lens and vitreous. At any rate this absorption process has nothing to do with the filtration angle, as pressure on the sclera and vitreous humor pushes forward the lens and closes the angle.

The most important consideration in the investigation of glaucoma is that of a correct diagnosis. If that is right no ornamental addition is called for.

It is senseless to call one and the same case glaucoma simplex to-day and simple glaucoma "with inflammation" to-morrow, as well as to talk of the possibility of every acute glaucoma becoming chronic, and every chronic glaucoma becoming acute. Doubtless there are differences between primary and secondary glaucoma, but one should use the latter term with great care, lest he designate totally different conditions by the same term, as has often happened in glaucoma simplex, and not infrequently occurs in inflammatory states.

[1] *v. Graefe's Archiv*, Bd. xix., 2, p. 87.
[2] *v. Graefe's Archiv*, Bd. i., 2, p. 101.

TRAUMATIC RUPTURE OF THE SCLERA IN THE ANTERIOR HALF OF THE BULBUS.

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(*With three wood-cuts and eight figures on plates xi.–xiii. of vol. xx., June edition.*)

Translated by Dr. J. M. Mills, New York.

THE attention which opthalmologists have bestowed, for a long time, upon the rupture of the foremost zone of the corium, has its motive especially therein that from these injuries changes are often produced similar to those caused by our cataract extraction. I gathered together from the literature[1] at least 144 cases, and, in addition, six from my own observation.

Among 97 cases of which I obtained accounts of the instrument of assault, 23 were caused by a blow from the horn of a cow or ox (see accompanying wood-cuts; Figs. 1 and 2, ends of cow horns; Fig. 3, of an ox horn), 19 by a blow or fall against the edge of some irregular object, 14 by a thrust or blow with the end of a wooden or iron bar or other similar instrument, 8 by a blow with the fist, 6 by a thrust or pressure of a finger against the eye, 1 by a push against the outstretched hand, 9 by pieces of wood flying into the eye, 3 by stones or earth-clods thrown into the eye, 4 by a blow from a horse's hoof; 3 were struck with a whip; 1 by a knock against the door-knob; 1 each by a blow with a hammer, knuckle-duster, rapier, and wand.

[1] In the older literature (beginning and first half of the present century) are found intimations of cases belonging to this class, though their complication with aphakia was not recognized, etc., etc.

The uniformity, just mentioned, in most of the cases, renders superfluous the presentation of more detailed histories.

Notwithstanding the large number of individual observations, the explanation of the occurrence of the indirect rupture of the sclera is still a matter of dispute. Whereas until a short time ago the opinions of ophthalmologists were divided between the theories of Manz,[1] Schröter,[2] and v. Arlt's[3] theory of flattening, now the latest investigator in this region, Hughes, endeavors to give this problem a purely mechanical solution.

a *b* *c*

FIG. 1. FIG. 2. FIG. 3.

Twelve years ago (at the time of the appearance of the large hand-book of Gräfe-Sämisch) the pathologico-anatomical knowledge concerning our injuries was exceedingly meagre; and although since then it has been greatly enriched by the excellent contributions of Alt,[4] Treitel,[5] and Schäfer,[6] they must still be considered deficient, as the examinations of the authors just mentioned consisted only of eyes that had to be enucleated on account of intense inflammation or

[1] Manz: "Zwei Fälle von Traumatischer Bulbusruptur."—*Klin. Monatsbl.*, Bd. iii. (1865), S. 170.

[2] Schröter: "Zur Entstehung der Traumatischen Scleralruptur."—*Klin. Monatsbl.*, Bd. iv. (1866), S. 242.

[3] v. Arlt: "Ueber die Verletzungen des Auges," e:c., Wien, 1875, S. 5–12.

[4] Alt: "Beiträge zur Patholog. Anatomie, Fall 3."—*Gräfe's Arch.*, xxxiii., 3, p. 26.

[5] Treitel: "Beiträge zur Patholog. Anatomie des Auges."—*Arch. f. Ophth.*, Bd. xxvi., 3, S. 109.

[6] Schäfer: "Aniridia et Aphiatraumatica."—*Arch. f. Ophth.*, Bd. xxix., 1, S. 13.

threatening sympathetic affection, whereas the anatomical condition of an eye that was cured (with retention of sight) has, until now, been wanting.

I hope to supply this want by the following somewhat more detailed description of the first case, and if I add thereto pathologico-anatomical communications of other eyes, injured in the same manner, but affected with severe inflammation, it is only because the contrasting of the different terminations of the same injuries would not be without profit, and also the number of autopsies appertaining thereto is still very small.

CASE 1.—The eye was removed on December 12, 1883, by my colleague, Dr. Purtscher, in Klagenfurt, from the body of a more than 60-year-old woman. Fifteen years before, she was injured in the right eye by a thrust from the horn of a cow. Dr. Purtscher, who kindly presented me with this specimen, noticed, on the body, the typical iris coloboma, upwards and inwards, but was not in a position to obtain further clinical details. The left eye of the subject was normal and was not enucleated.

The normally formed bulbus, after remaining several years in Müller's fluid, was further hardened by me in alcohol, then opened by an equatorial section, upon which the fluid part of the vitreous humor escaped. Upon examination of the anterior half of the bulbus, from within (Fig. 1), the absence of the lens is right away noticeable, instead of which a transparent diaphragm, consisting of the more consistent parts of the vitreous, covers the posterior surface of the iris. There is a defect in this, directed upwards and somewhat inwards.

The anterior half of the bulbus was imbedded in celluloidin for the purpose of microscopic examination, and a series of sections made parallel to the plane of the middle line of the coloboma.

An arched cicatrix occupies the place of the scleral rupture, which encircles the upper half of the corneal periphery.

The rupture lies, in its whole extent, in the scleral border of the cornea, beginning in the medial line, almost on the horizontal meridian, ending laterally somewhat above the same, and penetrates at its lateral end only the superficial

scleral layers, whereas otherwise it perforates every part. Schlemm's canal is obliterated. The cicatrix attains its maximum width somewhat medial from the vertical meridian. The cicatrix consists of non-vascular fasciculi of connective tissue that have mostly a meridional direction, are not particularly rich in nuclei, are partly straight and partly slightly undulating; the cornea is unchanged. The sclera adjacent to the cicatrix is somewhat thinned and more strongly arched. The conjunctiva is adherent to the cicatrix, but is not ruptured. The scleral cicatrix inclines obliquely, from behind—upward towards the front—and downward, its proximal edge deeper than the distal, and covered by the ciliary processes (Fig. 2, Plate xiii.). Not only the apices of the ciliary processes, but also the vitreous body, are connected with the scar. Somewhat posterior to the place of rupture there is in the scleral tissue, thickened by cicatricial bands, a cavity which contains the lens capsule, denuded of its epithelium and very much wrinkled. Back of the lens cavity, directly episcleral, a strip of pigmented tissue has healed into the wound, and can be recognized as a remnant of iris by its vessels and adherent uveal pigment. The corpus ciliare of the opposite side is normal. On the medial, as well as on the lateral side, the ciliary process and vitreous are more extremely incarcerated in the cicatrix. On the nasal side the iris, doubled up, is wedged in the scar (Figs. 3 and 4); at the periphery it is ruptured (iridodialysis). Near the lateral periphery of the chamber the rupture penetrates the superficial fasciculi of the sclera. Schlemm's canal is intact, whereas, on the contrary, the sinus of the chamber is altered in such manner that the processes detached from the root of the iris are drawn near to the inner wall of Schlemm's canal, but are not adherent. Thereby also the anterior triangular side of the corpus ciliare appears shortened. The foremost portion of the retina shows cystoid degeneration, whereas the posterior section, as well as the optic nerve, are entirely normal.

Case 2.—M. Georg. Entered v. Jäger's clinic on Oct. 24, 1875. Twenty-three weeks previously the left eye was injured by a piece of wood flying against it. Six weeks after the injury

suffered intense and continuous pain. Thereupon began an inflammation in the right eye that, at the time of admission, was recognized to be a pronounced sympathetic iridocyclitis. The left injured eye was atrophied, soft, and without a lens; above the corneal margin was a swelling that contained the dislocated lens. Enucleation on October 6, 1875.

The anterior half of the bulbus, left to me, was cut in half by a vertical meridional section. The sclera in the region of the rupture is thinned, otherwise thicker than normal. The rupture, almost in its entire extent, passes through corneo-scleral margin, so that the edge of the scleral wound corresponds about to the insertion of the ciliary muscle, and Schlemm's canal is included in the rupture. Only at one end the rupture lies farther backward, and only the sclera is involved, the uvea remaining intact.

The cicatricial mass consists of tough, nucleose reticular connective tissue, which unites the lips of the wound and connects externally with a pouch, the empty bed of the dislocated crystalline body (Fig. 5, *cfl*), and internally with a pseudo-membrane lining the ciliary body (*cye*, *cye'*). The iris is partially imprisoned in the scar, partially detached and tilted back.

The ciliary body is infiltrated in every part by a very extensive inflammatory product. Below, the extremely swollen processes of the posterior surface of the iris come so close together that they almost touch, whereas, on the side of the rupture, they show a much less degree of turgescence. The fascicula of the ciliary muscle are pressed apart; the form of the triangle of the muscle is changed at the apex by the enlargement of the angle; the nuclei on and between the fasciculi of the muscle are considerably increased. The tissue between the muscular and pigment layer exhibits an almost continuous deposit of round cells, in which hardly anything of the matrix can be distinguished. The layer of cylinder cells on the pars nonplicata corp. cil. is partly detached from the pigment-layer; the individual cells are considerably elongated, and have grown out into fibre-like formations. On the inner surface of the corpus ciliare and on the posterior surface of the

iris are lodged masses of exudation that contain in the delicately fibred matrix numerous round and spindle-cells. The cyclitic membrane sends off shoots toward the axis of the eye.

The infiltration of the corpus ciliaris extends to the foremost part of the choroid, which is considerably thickened and appears to be permeated by round cells, that in places are massed into regular pus-nodules. Partly behind the iris and partly near the margin of the pupil, projecting into the anterior-chamber space, are extensive fibrinous coaguli, which contain numerous leucocytes in the marginal portion, but only a few in the middle. A similar very large coagulum lies eccentric, near to the corpus ciliare of the ruptured side.

There is not even a trace of the zonula Zinnii visible. The anterior section of the retina has become adherent to the previously mentioned layer of exudation; it is detached and very much wrinkled, its structure materially altered by hypertrophy of the basilar fibres, and by numerous larger and smaller hemorrhages that have undergone degeneration.

CASE 3.—This eye was enucleated at v. Jäger's clinic in Vienna. Unfortunately there is no account of the nature of the injury or of the clinical condition.

The bulbus was cut in half by horizontal meridional section, and has a sagittal diameter of 24 *mm*, and a horizontal of 21 *mm*, the other a sagittal diameter of 24 *mm*, and a horizontal of 23 *mm*.

Medially on the corneal border is a rupture of the sclera plugged by a cicatricial mass, the edges of the wound being very much curved in (Fig. 6). A capsule of connective tissue is connected externally with the cavity of the dislocated lens (*cfl*). The iris is applied to the posterior surface of the cornea. The rupture is situated exactly on the corneo-scleral margin, comprising almost half of the conical periphery. Internally the rupture is plugged by new-formed connective tissue connected with the ciliary body, and the adjacent part of the detached retina, constituting a very much wrinkled pseudo-membrane that is not sharply separated from the cicatrix; it is permeated by numerous

hemorrhages from new-formed vessels, and contains accumulations of pigment together with gland tube-like proliferation products of the cylinder-epithelial layer. Pupil obliterated. The remnant of anterior chamber is empty.

The corpus ciliare is infiltrated with leucocytes. The cord of the optic nerve is thinner, the nerve-fibre bundle narrower and more granular. The connective-tissue septi are thickened and very rich in nuclei. The intermarginal space (particularly distinct at the bulbar extremity) is enlarged; the arachnoidal sheath, thickened.

CASE 4.—The right eye of L—— G——, on January 10, 1885, injured by the horn of a cow. Immediate blindness. Enucleation twenty-four days later. Globe atrophic. In the inner-upper scleral border is a rupture 10 *mm* long, 2–4 *mm* wide, curving 3–4 *mm* backward. Cornea flat. Ciliary body and iris absent at the place of the rupture.

Microscopic.—The lumen of Schlemm's canal, on the side of the rupture, is destroyed. The retina is in the wound. Cyclitic membrane. Corpus ciliare more atrophied than inflamed. The adjacent choroid thickened by contraction and dense leucocytic infiltration. The optic nerve presents, at its intraocular extremity, evidence of inflammation; in its orbital portion, the picture of atrophy.

CASE 5.—P. B. sixty-six years old, l. e. hurt with the finger, intense pain, total blindness. Rupture of the sclera near the medial part of the cornea. Eye removed thirty-three days later. (See Fig. 7, Plate xii. and Fig. 8, Plate xiii.)

Around the rupture there is considerable hemorrhage (*sg.* Fig. 8.) in the adjacent structures. Wound is not cicatrized. Schlemm's canal obliterated. Retina and choroid detached. Lens absent.

CASE 6.—Z. M., forty-eight years old, on May 21, 1881, l. e. struck and became blind immediately. Great pain for four weeks. In seven weeks right eye inflamed. Iritis (sympathetic?). Medially and 1 *mm* behind the cornea, a subconjunctivo-scleral rupture 8 *mm* long, 3 *mm* wide. Eye enucleated two months after the injury.

Lens absent; also the iris near the wound. Choroid detached. Ciliary body protruding through the scleral wound. Choroid infiltrated with pus cells. Only the first eye recovered and probably preserved good sight permanently. In the subconjunctival rupture the iris and ciliary processes were incarcerated. In cases 2 and 3 severe irido-cyclitis occurred despite the subconjunctival rupture of the sclerotic. Yet small lacerations of the conjunctiva, not discoverable by the anatomical examination, may have existed.

If the scleral wound is made an open one by a simultaneous rupture of the conjunctiva, then are the chief conditions for an infection present, but when occasionally such cases have a favorable course, then is it due either to happy chance or to the asepsis used which kept the phlogogenic agencies from the eye.

It is much more difficult to understand those cases in which an eye, with rupture of the sclera having induced a sympathetic affection of the severest form, itself, however, remains entirely or almost free from inflammation. Such observations are communicated by Schrag, Jacob, Bresgen, Arlt, and Manolescu.[1]

Also these authors will accommodate themselves to the microphytic theory, but it remains for the future to prove if the explanation, given by Deutschmann,[2] the exact and

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[1] Schrag ("Einige Fälle von Rupturen der Sclera und Choroidea," J. D., Leipzig, 1870, Fall 3). Right eye: Partly open, partly sub-conjunctival situated scleral rupture. Lens luxated into the vitreous. S. with + ½ Jg. 5. Left eye: Sympathetic irido-cyclitis, S. Jg. 6.

Jacob ("Some Varieties of Luxation of the Crystallin." *Brit. Med. Jour.*, March, 1870, S., 226). Sub-conjunctival luxation of the lens upward. S. retained. The other eye has been lost by sympathetic inflammation.

Bresgen ("Zwei Fälle von sympathisch. Erkrankungen." *Wiener med. Wochenschr.*, 1878, S. 1197). Right sub-conjunctival scleral rupture. Lens luxated into the vitreous. S. $\frac{20}{50}$. Left sympathetic iritis with opacities of the vitreous, healed without enucleation of the injured eye and with retention of the normal S.

Manolescu ("Aniridie et aphakie traumatiques." *Arch. d' Opht.*, 1885, Bd. v., No. 3, Mai–Juni, S. 227). S in injured, = fingers in 9 meters. In the other eye sympathetic irido-choroiditis, that has run its course. S, after repeated operations, fingers in 50 *cm*.

Besides these there are among the 120 cases still 5 in which the injury had caused sympathetic ophthalmia, of these there were 4 in which the scleral rupture was sub-conjunctival.

[2] Deutschmann: "Ophthalmia Migratoria," Hamburg, 1889, S. 133.

circumspect originator of the migration theory, is more than a mere digression from the facts. "On the other hand it is just in this acceptance of this infection theory, not excluded, that in rare instances the cells of the organ wage a successful war with the micro-organisms, that have entered, for the preservation of the eye. So that the first affected eye, though unable to prevent the rapid propagation of the germs along the optic nerve apparatus, may nevertheless finally render the germs it contains harmless, and thereby preserve a high degree of valuable functional power; whilst the eye secondarily affected, succumbs to the micro-organisms with which it is infiltrated."

The deviations from the typical positions and direction (circumcorneal) of the rupture are as remarkable as they are rare; for instance, v. Stellwag[1] and Fano[2] describe a rupture that, following the direction of a meridian and laterally situated in the anterior scleral zone, nevertheless permitted the lens to pass under the conjunctiva, and in one case of Schröter's,[3] joined on to the posterior end of an indirectly produced wound (having also a meridional direction), was a second angular rupture—deviating from the former and forming with it a flap.

That the scleral rupture in the large majority of all the cases belongs to the upper periphery of the bulbus has been a well-known fact for a long time.

From the grouping of the material published we ascertain that the situation of the rupture was above 36 times, above and inwards 21 times, above and outwards 11 times, inwards 20 times, inwards and downwards twice, outward 8 times, and outwards and downwards once.

The length of the rupture was uniformly so extensive that it spanned nearly $\frac{1}{3}-\frac{1}{2}$ of the corneal periphery; clinical experience also teaches that the rupture exceeds in length, on the average, a cataract section situated in the limbus.

The situation of the rupture, as described, necessitates almost constantly the participation of the iris in the injury;

[1] v. Stellwag: *Ophthalmologie* 1856, Bd. ii., 1, S. 468, Anm. 218.
[2] Fano: "Lussazione sotto congiuntivale del cristallino," *Annal. di ottalm.*, Bd. ix., 3 u. 4, S. 364.
[3] Schröter, *l. c.*, Fall 4.

this is almost constant, as in the published statistics there are only two cases in which the lens had escaped through the rent of the foremost scleral zone without destroying the circular form of the pupil.

The contrast to the intact-remaining iris is offered by the complete aniridia.[1] Among 83 cases it is mentioned 17 times, with exact information of the condition of the iris; frequently there remained besides very insignificant remnants of the iris. In only two reports is the dislocation of the completely detached iris under the conjunctiva mentioned (Haenel[2] and Homburg[3]); more frequently the iris escapes through the conjunctival wound at the same time with the lens.

That a true coloboma of the iris occurs is proven by the first communicated autopsy; the shanks of the coloboma, from which the border of the sphincter is absent, are recognized as the edges of the rupture by their covering with hyaline tissue, that is foreign to the normal pupillary border.

The section torn from the continuity of the iris-breadth was at the same time detached from its ciliary insertion and partly healed in the pocket-like protrusion of the cicatrix and was considerably shrunken.

In an isolated observation of A. v. Gräfe,[4] a relatively small vertical piece, broken from the equatorial region of the lens, escaped through a scleral rupture under the conjunctiva, whilst the principal mass of the lens system remained intraocular and in its normal position.

I am much less inclined to harbor the illusion that my few suggestions have made entirely clear the mechanism of the rupture of the sclera, as I myself still foster the conviction that the final decision rests with the experiments employed.

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[1] (a) Dixon, James, *The Lancet*, Nov. 27, 1852, cit. n. Massie, Étude, etc. (b) Bericht der Arlt'schen Klinik von Tetzer, Becker und Rydel, 1866, *Wiener med. Presse*, 1866, S. 1205, Fall 2.

[2] Haenel, Jahresber. der Gesellsch. f. Natur- u. Heilkunde in Dresden, 1881 S. 30.

[3] Homburg, "Statistik und Casuistik der Augenverletzungen" (aus Hirschberg's Material), *Inaug.-Dissert.*, Berlin, 1883, Fall 376.

[4] A. v. Gräfe, "Beobachtung einer partiellen Dislocation der Linse unter die Conjunctiva," *Arch. f. Ophth.*, Bd. iii., 3, S. 369.

Explanation of the Figures 1–9.

Fig. 1 (Case 1). Inner view of the anterior half of the bulbus. The vitreous remnants are not drawn. Twice magnified. *c*, cornea; *ci*, cicatrix; *i*, iris; *mco*, *mco'*, medial and lateral coloboma margin; *os*, ora serrata; *pc*, processus ciliaris; *s*, sclera.

Fig. 2 (Case 1). Section parallel to the middle line and somewhat lateral to the medial border of the coloboma. Verrick, obj. 2, ocul. 1. *c*, cornea; *ci*, cicatrix; *cj*, conjunctiva; *cl*, capsule of lens; *cv*, vitreous; *i*, episcleral dislocated piece of iris—fragment of iris in the conjunctiva; *mc*, musculus ciliaris; *pc*, processus ciliaris.

Figs. 3 and Fig. 4 (Case 1). Section somewhat medial from the middle line of the coloboma and parallel. Fig. 3 magnified. Fig. 4, Verrick, obj. 2, ocul. 1. *c*, cornea; *ci*, cicatrix; *cj* and *cj'*, conjunctiva; *cc* and *cc'*, corpus ciliare; *cv*, vitreous; *i* and *i'*, iris; *md*, Descemet's membrane; *pc*, processus ciliaris.

Fig. 5 (Case 2). Vertical meridional section magnified. *c*, cornea; *cc* and *cc'*, corpus ciliare; *ci*, cicatrix; *cj* and *cj'*, conjunctiva; *cye* and *cye'*, cyclitic exudation; *cfl*, fibrous capsule of the dislocated lens; *fc*, fibrinous coagulum; *i* and *i'*, iris; *md* Descemet's membrane; *r*, retina; *s* and *s'*, sclera.

Fig. 6 (Case 3). Horizontal meridional sections. Highly magnified. *c*, cornea; *cc* and *cc'*, corpus ciliare; *ci*, cicatrix; *cj* and *cj'*, conjunctiva; *cfl*, fibrous capsule of the dislocated lens; *cl*, structureless capsule of the lens; *ecy*, cyclitic pseudo-membrane; *i* and *i'*, iris; *r*, retina; *s* and *s'*, sclera; *sa*, anterior synechia; *spch*, suprachoroid.

Fig. 7 (Case 5). Inner surface—view of the upper end of the rupture—magnified twice. *c*, cornea; *ci*, conjunctiva; *md*, edge of Descemet's membrane; *rs*, scleral rupture; *s*, sclera; *ss* scleral groove; *sw*, scleral eminence.

Fig. 8 (Case 5). Section through the middle of the rupture and perpendicular to the course. Fifteen times magnified. *cj*, conjunctiva; *lcj*, limbus of the conjunctiva; *rcj*, conjunctival rupture; *eps*, episcleral tissue; *i*, iris; *mc*, musculus ciliaris; *pc*, processus ciliaris; *s*, sclera; *sg*, blood.

ON PAPILLOMA OF THE CONJUNCTIVA.

By Dr. SIGMUND FUCHS.

FROM THE EYE-CLINIC OF PROF. ERNST FUCHS IN VIENNA.

Translated by Dr. WARD A. HOLDEN, New York.

(*With plate xiv. of vol. xx., German edition.*)

THE tumors which I shall describe here belong to the general group of proliferation tumors or growths (Virchow), and to that special class which genetically at least are composed chiefly of connective tissue.[1]

Commonly called papilloma (Krämer), the more fitting name is fibroma papillare (Virchow). In many cases the epidermoid or epithelial strata make up no small component of the tumor, particularly in those papillary fibromata which develop as a plate or skin-like mass of connective tissue.

In some of these cases the connective tissue forms so small a constituent of the tumor and the epithelium so predominates that the fibrous character of the growth might be disputed, but a study of the genesis of the tumor always shows that in reality it belongs to the class of fibrous growths, and the small amount of fibrous tissue present is due to the fact that there has been a retrograde metamorphosis or an atrophy caused by the excessive development of the epithelial constituent.

Papilloma may develop in any portion of the conjunctiva, and since all portions of the conjunctiva do not possess papillæ, it cannot always be regarded as a simple hypertro-

[1] Virchow, "Geschwülste," vol. i., p. 287 *et seq.*

phy or as an excessive development of normal papillæ. In the initial stage all these papillary growths appear as a small round knob due to excessive development of the superficial tissue; vessels push into the connective-tissue centre of the little bud and at the same time the epithelial layer grows thicker. New sprouts appear on the bud, and become vascular, forming finally a branched papilla. The papillomata which spring from the tarsal conjunctiva often assume a cauliflower appearance with a very wide base, since the process just described takes place at a number of different points. Similar forms of less extent are seen on the plica semilunaris. Those developing on the bulbar conjunctiva and in the fornix are, on the contrary, branched pediculated vegetations with a papillary surface, the whole having rather a polypoid form.

What has just been said will explain the similarity of papillomas of the conjunctiva to the pointed condyloma, and will show that it is akin to the warty formations of the dermatologists.

Fibroma papillare of the sclero-corneal border in its initial stage can hardly be distinguished clinically from epithelioma, but when it has reached a certain development and overlaps the cornea, the involvement or non-involvement of the substance of the cornea is a diagnostic sign of great value. For a papilloma which overlaps the cornea may be lifted up and pushed back to the conjunctiva, *but an epithelioma will infiltrate the corneal substance and rest immovably on it.*

Another valuable point is the enlargement of the neighboring lymphatic glands, which, of course, never occurs with papilloma. In all doubtful cases histological examination is required for an accurate diagnosis.

The papillomata of the limbus are distinguished from so-called spring catarrh by their softness and their papillary surface. The individual papillæ have always a small base and a broader extremity and are somewhat movable. Spring catarrh is characterized by photophobia, by its regular recurrence in the spring, and by the fact that it only affects children and adolescents.

No certain causes for the development of papilloma are known; I would refer the reader to the paper by Elschnig[1] for information on this point.

The tumors are benign, although inclined to recur if not radically removed. In no case observed did the growth become sarcomatous or carcinomatous, and the microscopic examinations never revealed evidences of any such change.

The only treatment is the operative removal of the growth, together with the underlying conjunctiva, to guard against recurrence.

The literature on hard and soft fibroma has been collected by Elschnig.[2] An exquisite case of papilloma of the conjunctiva was reported by Magnus in *Zehender's klin. Monatsbl.*, vol. xxv., 1887, p. 384.

The material for these investigations was composed of eleven cases of papilloma seen in Prof. E. Fuchs' clinic in the course of the years 1887–1888; two of these cases were observed for a considerable time, the nine others occurred in out-patients. Details regarding age of patient, situation of growth, duration, etc., were not kept in the latter cases. Histologically they were all similar.

CLINICAL REPORTS.

CASE I.—M. Hansl, æt. 28, presented himself in July, 1887, with a small tumor at the left internal canthus which was removed. In August he noticed a return of the growth and in October the condition was as follows: The lower lid is slightly everted and rising above its margin is a finely papillary red neoplasm; a similar growth is seen at the middle of the free margin of the upper lid. The tumor spread over the tarsal conjunctiva of the inner half of the lower lid as a narrow stripe, and at the inner canthus extended over the fornix to the bulbar conjunctiva. The upper fornix was free from the new growth. The tarsal conjunctiva of the upper lid, in almost its entire extent, is covered with the new growth, which is here flatter and more compressed. The tumor ceases sharply at the conjunctival margin. The portion on the conjunc-

[1] Elschnig, these ARCHIVES, No. 3, 1889.
[2] *L. c.*

tiva is more granular. There is a small isolated tumor near the cornea.

October 29th, the tumor, together with the underlying conjunctiva, was extirpated, and the conjunctiva united by sutures. The wound healed by first intention, but caused a slight entropium of the upper lid. Nine months later a tumor 3 *mm* in diameter was found in the upper lid and removed. The conjunctiva was otherwise normal.

CASE 2.—I. Pollack, æt. 68, admitted with senile cataract. The conjunctiva of the right lower lid and the lower half of the bulbar conjunctiva was somewhat injected. From the lower outer quadrant of the conjunctiva bulbi springs a lobular tumor the size of a hazel-nut, of soft consistence, rose-red color, and pervaded with numerous small vessels. The tumor hangs from a small pedicle and spreads out over a considerable area. When the lower lid is retracted the tumor projects through the palpebral tissue and is only returned with difficulty. It was first noticed a year before. The tumor was removed with the scissors and the wound healed rapidly.

HISTOLOGICAL CONSTITUTION.

In all the cases examined the connective-tissue framework was poorly developed in comparison with the epithelial strata. The connective tissue stroma was most marked in Case I. It was of the ordinary type of fibrous tissue such as is seen in the connective-tissue portion of the conjunctiva. At many points it showed a reticular arrangement, and its meshes were filled with lymph corpuscles; in the individual papillary elevations of the tumor the connective tissue extended in parallel tracts, compressed in the pedicle and spreading out into a reticular structure in the papilla proper. In the smallest isolated portions of the tumor the connective-tissue tracts were irregularly arranged, and were composed of fine fibres and provided with a great number of blood-vessels, so that the connective tissue often presented itself as an adventitial sheath of the vessels. The epithelium covering the tumor made up its chief mass. Near the connective tissue it was mostly cylindrical in form, and above this the cells gradually assumed the pavement form.

Division by Fragmentation (J. ARNOLD).

Beside the mitotic figures which are seen in all stages, there are a series of figures which must be regarded as examples of Arnold's[1] type of direct fragmentation.

The variety of these forms observed and their irregularity warrant an accurate description. In all, the chromatic substance was degenerated into from two to six masses which stained well and showed no traces of fibrillar structure: the masses were often united, however, by fibrils, which stained well, or in case the fibrils were broken, the separate masses showed fibrillar processes. These forms were not seen frequently near the vessels, but were seen particularly in cells which lay in the superficial layers of the epithelium, among cells with unchanged nuclei and cells showing mitotic changes. The nuclei of the individual cells were of inconstant and irregular form. The nuclear membrane had always appeared and the structureless chromatic masses lay distributed through the substance of the cell.

After a very detailed description of karyokinesis according to modern views (which as belonging to general histological technology have been admitted in the translation), the author comes to the conclusion that the course of the mitotic division in the epithelial cells of papilloma is exactly of the type which is common in many of the lower vertebrates.

PAPILLOMA OF THE PALPEBRAL CONJUNCTIVA WITH PAPILLOMATOUS FORMATION OVER THE WHOLE SURFACE OF THE SKIN.

J. D., æt. 59, suffered in 1878 from hæmaptoë and an eruption on the skin with photophobia, lachrymation, swelling of the lids, and warty protuberances on the lids. In 1880 portions of the skin were covered with more or less prominent warty excres-

[1] Arnold, after extended investigations (see Waldeyer, *Arch. f. mikroscop. Anatomie*, vol. xxxii., 1888, p. 111), recognized the following forms of nucleus division: (1) Segmentation, direct and indirect; fragmentation, likewise direct and indirect. "If the nucleus split in an æquatorial or in a meridional plane, and the two parts, mostly of equal size, separate with an even surface, we have 'segmentation.' This is indirect when accompanied by karyokinetic changes, otherwise direct. In fragmentation the dividing surfaces of the daughter nucleus are irregular. Here also karyokinetic changes may be found (indirect fragmentation), or are wanting (direct fragmentation)."

cences, partly pointed, partly bulbous. The limits of the growths were not sharply marked. The eyelids and the neighboring skin were thickly covered with pigmented papillomata the size of a hemp seed and smaller, projecting 2–3 *mm.* The conjunctiva was also covered with small warts, larger toward the lid margin. The lids were thickened, darkly pigmented, and slightly reddened ; the cilia were for the most part wanting ; the muco-purulent conjunctival secretion formed crusts on the lids.

Therapy : one per-cent. solution of salicylic acid. The patient lost weight, complained of pain in the stomach and pain on pressure in the pyloric region. A profuse diarrhœa came on ; the patient became worse, and died Jan. 10, 1881. The necropsy showed carcinoma of the posterior wall of the stomach, endarteritis of moderate degree, shrunken kidney, papilloma of skin, marasmus senilis.

Pieces of skin taken from the cadaver were hardened in absolute alcohol, and imbedded in celloidin. The upper lid was cut in sagittal sections and stained with carmine, safranin, and Ehrlich's hematoxylin and eosin. The preparations so obtained showed the skin and mucous membrane of the lids to be covered with tufted finely papillary warts. On the conjunctiva of the lid toward the fornix the papillæ were hypertrophied but not branched. The connective-tissue stroma of each papilla was composed of finely meshed fibrous tissue containing lymphoid cells, and corresponding to that of the broad papillæ which are found in the conjunctiva of individuals suffering from long-continued conjunctival catarrh. The epithelium is a many-layered pavement-epithelium, with an underlying layer of cylindrical cells.

All these formations showed on their surface the characteristics of the mucous membrane from which they sprang. At the free margin of the lid the individual papillomata took on an epidermoid character. Each presented on its surface a layer of cells corresponding to the epidermis, and a subjacent connective-tissue layer rich in vessels and nerves, corresponding to the corium. The epidermoidal portion increased in thickness from the free margin of the lid toward the outer surface, and was seen to consist of two layers,

corresponding to the stratum corneum and the stratum germinativum of the true epidermis. The first layer is composed of several layers of flat scales without nuclei and taking the stain very faintly. The germinative layer, on the contrary, is composed in its deepest portion of a single layer of cylindrical cells resting immediately on the free surface of the sclera, while its superficial portion consists of polygonal prickle cells. The entire germinative layer is thickened, and the smaller papillomata are due to a proliferation of the cells of this layer. The mesoblastic portion of the individual elevations is composed of numerous conical formations—the papillæ corii. These are very vascular, contain numerous nerves, and pass over into a finely fibrillar non-fatty subcutaneous connective tissue, identical in histological structure with the connective-tissue basis of the normal skin of the lids. In the larger papillomata which show a marked development of the mesodermic connective-tissue layer, these subcapillary tracts of connective tissue have taken part in the structure of the individual excrescences. Some of the papillæ show new hair formation.

The ætiology of this case is not clear; no specific micro-organisms were found in the tissues. The effect of therapeutic remedies could not be studied, because of the lethal ending, which came soon after the patient's admission to the clinic.

I have found but one analogous case in the literature, reported by Lang.[1]

Explanation of the Figures on Plate XIV.

Fig. 1. Axial longitudinal section through a bud of the papilloma in Case 2. Slight development of the connective-tissue stroma, marked development of the epithelial covering. Ob. 2, Oc. 3. Reichert, tube drawn out.

Fig. 2. Sagittal section through the entire thickness of the upper lid of the case of general papillomatosis. Ob. 2, Oc. 3. Reichert, tube drawn out.

[1] Lang. A case of papillomatosis universalis (from the dermatological clinic of Prof. Lang at Innsbruck).—*Vierteljahreschr. f. Dermatologie u. Syphilis*, 10. Jahrg., 1883, p. 337.

RETINITIS DIABETICA.

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(*With one Wood-cut.*)

RETINITIS diabetica has hitherto been the name of retinal inflammations depending upon *diabetes*, without signifying any peculiar disease which could be recognized by its definite and characteristic appearance. The various forms in which it had been seen and described were considered very much the same as those of albuminuric retinitis. Thus Noyes, in his text-book,[1] published in 1890, says that "the features of both are to a great degree identical."

In Loring's work,[2] which has just been published, we find the statement that "the diabetic form of retinitis offers but little which is distinctive of presence of sugar in the urine. As a rule, however, there is more hemorrhage and less exudation or white plaques than in retinitis albuminurica, and the spots are similar and not so apt to be situated in the macula, or grouped in a zonular manner around the papilla." Gowers is scarcely more accurate. In the latest edition of his work on ophthalmoscopy[3] he describes the retinal changes as "bearing considerable resemblance to those of albuminuria, and still greater resemblance to those seen in some cases of pernicious anæmia. . . . White spots of degeneration are frequently present, commonly of moderate size, scattered over the fundus. . . . They differ from

[1] "Diseases of the Eye," p. 568.
[2] "Vol. ii., p. 152.
[3] Amer. edition of 1890, p. 228.

the patches of albuminuric retinitis in *shape*, having less tendency to assume a circular form; in *color*, having a more dingy shade of white, and in *grouping*, the star around the macula being seldom seen, although there is a tendency for the spots to be arranged in the form of incomplete rings."

Other American and English authorities are much less definite in their descriptions, and German authorities are equally unsatisfactory.[1]

That characteristic differences do exist between diabetic retinitis and other forms of retinal inflammation was pointed out several years ago by Prof. Hirschberg,[2] of Berlin. In a more recent article[3] he demonstrates that there are typical forms of diabetic retinitis, which present characteristic appearances. The number of cases upon which he bases this study is very large, comprising twenty-five cases, "which is a greater number than has hitherto been published by any single observer." He distinguishes three kinds of retinal disease that occur in diabetic patients. "First, a very characteristic inflammation of the central part of the retina with small, bright spots, and usually fine spots of blood. Second, hemorrhages of the retina, with the consequent inflammatory and degenerative changes, including hemorrhages into the vitreous, hemorrhagic infarction of the retina, and hemorrhagic glaucoma. Third, rare forms of retinal inflammation and degeneration, whose connection with the constitutional disease yet remains to be demonstrated." There are, of course, intermediate forms, presenting the characteristic features of more than one type.

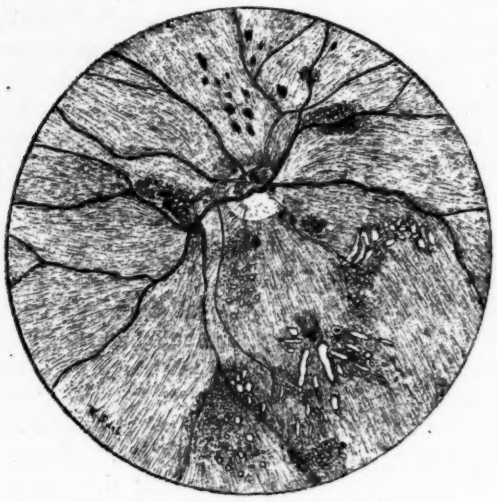
In the first class we find small, white, shining spots in the central part of the retina. These lie in the retinal tissue. In the macular region they are most marked, often forming little semicircles or curved streaks, with minute indentations along their margins. They may become confluent and form long streaks, though they rarely present the stellate form of albuminuric retinitis. A few bright spots may be seen scat-

[1] See Hirschberg, *Deutsche med. Wochenschr.*, 1890, No. 51.

[2] "Sehstörungen durch Zuckerharnruhr," *Deutsche med. Wochenschr.*, Nos. 17 and 19, 1887.

[3] "Ueber diabet. Netzhautentzuendung," *Deutsche med. Wochenschr.*, 1890, Nos. 51 and 52.

tered over the retina to the nasal side of the papilla. They never cause any disturbance of the retinal pigment. Fine hemorrhages may be visible throughout the region affected and beyond it, in the form of dots, small spots, and fine streaks. In these cases, the papilla is not altered either in its color or its margin; there is no diffuse haziness of the retina, nor are there any visible changes in the blood-vessels, such as variations in calibre. Large opacities of the vitreous are not found in this form. It is this type that is pathogno-



RETINITIS DIABETICA.

monic of diabetes. The disease occurs in persons of from forty-five to sixy-five years of age, who have commonly suffered with diabetes for a long time, often for several years, occasionally though to such a slight extent that their general disease remained undetected before the eyes became affected. It usually develops gradually, and always affects both eyes. The visual disturbance is due to central scotoma. Vision ranges from $\frac{1}{2}$ or $\frac{1}{3}$ to $\frac{1}{10}$ or $\frac{1}{20}$, but not to complete blindness. It may improve slightly, but usually will either remain stationary or progressively diminish. There may be

lenticular opacities, and the patient may have paresis of the accommodation at the same time.

Hirschberg believes that these white spots owe their origin to changes in the finest blood-vessels; as mentioned above, the larger vessels appear normal. Their histology is not known.

During the last year a case quite typical of this form came under my observation, and the patient, a lady sixty-four years of age, who had been suffering with diabetes for several years, it having been looked for and detected by her family physician as the cause of pruritus vulvæ of long standing. Albumen has never been found in the urine. Her general condition had not suffered much during these years, though the patient had lost some weight. Her vision had become affected within two to three years, and was found to be: L E, + 2.5 D sph V $\frac{5}{10}$; R E, + 2.75 D sph V $\frac{6}{6}$ (June 16, 1890), the impairment being due to central scotoma, as is always the case in this form. The ophthalmoscopic examination shows a few peripheral striæ in both lenses, but otherwise the media were perfectly clear. The retina presented the typical form of diabetic retinitis described above, and shown in the drawing of the upright image of the retina of the left eye, which was slightly more affected than the right. There are the intensely bright, white spots arranged around the macula in streaks and lines, and, perhaps, approach the stellate figure more than is commonly the case. There are other spots of degeneration, less marked and finer in form, farther from the macula, though few reach beyond the papilla. Scattered hemorrhagic spots are seen among the white spots and to the nasal side of the papilla. The papilla is quite clear, of normal color and sharply defined. There were no abnormal variations in the calibre of vessels.

I have not been able to repeat the examination since then, but have learnt that her vision has become slightly worse. The treatment has been purely dietetic.

A CASE OF PULSATING EXOPHTHALMOS.

By P. B. WING, M.D., Tacoma, Washington.

Patient, a young man, age about 22, occupation laborer. In August, 1889, he fell some distance, striking on his head, was taken up in an unconscious condition and carried to the hospital. He remained in this condition for twenty-four hours and was in the hospital about ten days, and was discharged apparently well.

In December following, about five months after the injury, he noticed that the right eye began to protrude; it progressed slowly until the following June, when his physician, Dr. Beardsly, came with him to my office. At that time the eye protruded so much that the lids could with difficulty be closed over it. The conjunctival vessels were large and tortuous. Vision about $\frac{20}{200}$. There was a distinctly pulsating tumor at the upper and inner angle of the orbit. There was not at that time and never had been any pain or soreness. By pressure the eye could be made to partly recede. Pressure upon the common carotid artery caused the pulsations to cease.

My diagnosis was pulsating exophthalmos, and I advised ligation of the common carotid as the operation offering the best results. I have no doubt that there had been a fracture at the base, passing across the cavernous sinus and causing a communication to exist between it and the internal carotid artery, thereby forcing arterial blood directly into the ophthalmic veins, causing the great distension and strong pulsation; in fact, it was a tumor composed of pulsating varicose ophthalmic veins.

The patient was again sent to the hospital and the common carotid ligated by Drs. Beardsly and Wintermute. The pulsations stopped at once and never since returned. The eye began to gradually sink back into its place, and to-day, fourteen months since the operation, the protrusion is not noticeable except upon

very close observation. The vision is $\frac{20}{40}$ +. Patient has been at hard labor for the past eight or nine months.

I have reported this case for three reasons—it is the only one I have ever seen, it made a fine recovery, and because I want some well-informed brother [1] to give us some statistics in regard to its frequency and the results obtained by the various operations for its relief.

[1] The fullest description of pulsating exophthalmos, with tabulated records of 106 cases, is by H. Sattler in Graefe-Saemisch's "Handbook of Ophthalmology," vol. vi., pp. 745 to 1074. One of the most brilliant cases is that by Dr. E. Grüning, described in these ARCH., vol. v., p. 40, in which both eyes were protruding and totally blind. Perfect recovery with immediate restoration of sight by ligation of the left common carotid. In a case of the writer's, a temporary cure was effected by ligation of the carotid, a permanent cure (of the relapse) by extirpation of the aneurismal varix of the orbit (these ARCH., vol. xii., p. 201). Ligation of the carotid cures the majority of cases.—H. K.

PRESSURE-INUNCTION IN THE TREATMENT OF CERTAIN CHRONIC HYPERPLASTIC CONDITIONS OF THE EYE.

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I HAVE ventured to propose the term *pressure-inunction* to describe a form of treatment involving mercurial inunction combined with more or less continuous pressure.

The analogy presented by a chronic inflammatory hyperplasia of the synovial and fibro-cartilaginous structures of the knee-joint to a chronic hyperplastic condition of the cornea, sclera, and vitreous body suggested to my mind the idea that, if mercurials combined with pressure could be of service in the one case, it might also be found useful in the other. I would urge in support of this contention that the selective power of drugs is well known, and that it is undeniable that certain diseases prefer certain tissues as a local habitation. I would further point out the developmental relationship between cartilage, fibrous tissue, sclera, and conjunctiva.[1] Are they not all derived from the mesoblast? and is it not admitted that a pathological relationship exists between tissues of the same developmental origin?

Arguing on these premises I have for many years carried on a series of observations, and have had ointments of various strengths and of different preparations of mercury compounded, and have arrived at a standard ointment made of

Calomel	gr. xxx.
Vaseline	ʒi.

[1] "Eierstock und Ei," Waldeyer; "Entwicklungsgeschichte des Menschen," Kölliker.

very carefully rubbed up. It is applied freely to the closed eyelids, and, in cases of corneal opacity, a small piece is inserted in the conjunctival sac. A pad of absorbent cotton is then applied and over it three or four turns of a good elastic flannel bandage. This dressing is worn from two to three hours twice daily.

The cases in which I have found most benefit from this treatment are those of corneal opacities of a dense type, interstitial deposit, plastic deposit on the lens from iritis, opacities of the vitreous, and some obstinate cases of scleritis. The improvement is slow and the treatment must be persisted in for some months. I have never seen mercurialization from this treatment. It will be found that some persons will bear a stronger ointment than the standard one, in others it causes too much irritation and must be weakened or intermitted. I have occasionally injected five minims of a 2-per-cent. solution of common salt beneath the ocular conjunctiva after the method of Brudenel Carter, and have intermitted pressure-inunction for a few days. The hypodermic injection of sodium chloride should not be repeated oftener than once in three or four days, and the initial dose should not exceed two drops.

While this method of treatment is by no means infallible, I still have been able to obtain results which I would not have hoped for under other circumstances.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FOURTH QUARTER OF THE YEAR 1890.

By St. BERNHEIMER, Heidelberg; C. HORSTMANN and P. SILEX, Berlin.

WITH THE CO-OPERATION OF

S. M. Burnett, Washington; Dantone, Rome; Hirschmann, Charkow; E. Marckwort, Antwerp; P. v. Mittelstädt, Metz; L. Werner, Dublin; C. H. A. Westhoff, Amsterdam; Schiötz and Ole Bull, Christiania, etc.

Translated by Dr. Ward A. Holden, New York.

Sections I.–V. Reviewed by Dr. St. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

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405. Norton, Geo. S. An analysis of 1,022 eyes examined under atropine. *Jour. Ophth., Otol., and Laryngol.*, Oct., 1890.

406. Suarez de Mendoza. L'audition colorée. *Soc. d'Opht. de Paris*, No. 4, 1890.

407. Sebrenikowa, Frau. Ophthalmological observations at the government hospital in Perm, during the year 1889. *Wjestnik Ophth.* No. 6, 1890.

408. Motschulsky. A case of complete cyclopia. *Russkaja medic.*, No. 43, 1890. (Description of a preparation in spirits belonging to the Society of Physicians in Kertsch.)

409. Haab, O. Pathological anatomy of the eye. Ziegler's "Text-book of Special Pathological Anatomy," sixth edition.

410. SCHWERSENSKI, G. On the changes in the periphery of the fundus. *Inaug. Dissert.*, Leipsic.

411. LORENZ. On the proper construction of clinics. *Centralbl. der Bauverwaltung*, J. 1889–1890, Berlin.

NORTON (405) tabulates carefully 1,022 eyes examined under atropine. He finds that the axis of the cylinder in astigmatism sometimes changes under atropine, in some instances as much as 45°, showing an unequal contraction of the ciliary muscle. Since he has been using the ophthalmometer he finds the use of atropine less necessary. As a guide in general to the use of atropine, he takes the fact that the glasses which have been ordered without atropine are unsatisfactory. BURNETT.

SEBRENIKOWA (407) reports 3,055 cases with 780 operations, 126 being extractions, 48 without iridectomy. She does not favor the latter method of operating. HIRSCHMANN.

HAAB (409), in his pathological anatomy of the eye, gives in a condensed form a general and very accurate description of the most important pathological changes. The careful drawings are of value, as is also the bibliography at the end of the chapters.

SCHWERSENSKI (410) in his dissertation spoke of the frequence with which the peripheric changes in the fundus, described by Magnus, were observed, and of the relative number of these cases which were to be ascribed to old age, sex, refraction, and inflammation. In general he often found these changes to have a certain etiological connection with senescence and with inflammation ; he regarded them also as characteristic for myopia, and in this case probably due to mechanical causes, such as stretching and tearing. He could not speak definitely as to their relation to hyperopia.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

412. DE SCHWEINITZ, G. A case in which the effect of homatropine upon the accommodation was unusually prolonged. *Ophth. Rev.*, vol. ix., p. 353.

413. MADDOX, E. The investigation by the rod-test of paresis and paralysis of the ocular muscles.

414. BERRY, G. Note on the metre-angle in latent and manifest muscular deviation. *Trans. Ophth. Soc. of the United Kingdom*, vol. x., p. 190.

415. BISSELL, E. F. Note on the shadow-test. *Jour. of Ophth., Otol., and Laryngol.*, Oct., 1890.

416. KOLLER, CARL. Elimination of the cornea and the effect upon the refraction of the eyes. *Amer. Jour. of Ophth.*, No. 7, 1890.

417. SIMI. Posterior sclerotomy. *Boll. d'ocul.*, vol. xii., p. 22.

417a. JAYS. Mesure de la convergence dans la vision binoculaire. Choix d'unité de convergence. Série métrique des prisms en oculistique. *Soc. d'Opht. de Paris*, Oct., 1890.

418. LEROY. Influence de la distance de l'observateur dans la mesure des amétropies à l'image droite. *Rev. génér. d'opht.*, No. 10, p. 433, 1890.

419. DRANSART. De la suspension dans le nystagmus des mineurs et la neuro-retinite. *Jour. d'ocul. du Nord de la France*, p. 38, 1890.

420. DE WECKER. Résection et arrachement du nerf optique. *Soc. d'Opht. de Paris*, 1890.

421. BUCHNER, H. The chemical irritability of leucocytes and their relation to inflammation and suppuration. *Berliner klin. Wochenschr.*, No. 47.

In DE SCHWEINITZ's (412), case after using homatropine twice, the accommodation was not wholly restored at the sixth day.

WERNER.

MADDOX (413) describes his glass-rod test in detail. He uses with it a scale either in degrees or in metres. With this test latent divergence may be shown, which could not be discovered in any other manner. WERNER.

BERRY (414) found the actual association between accommodation and convergence in cases of squints to be given by the measurement of the metre-angle, the half of the angle of deviation being taken. WERNER.

BISSELL (415) has used the shadow-test for some time and testifies to its utility. It is easy to use, and by it results may be obtained where other methods fail.

After a detailed description of the optical phenomenon of Bellarminoff's method of making the fundus visible by eliminating the refraction of the cornea, KOLLER (416) described his modification of this method. In place of a plane glass plate, he uses a glass shell like an artificial eye, but with the convex outer surface ground plane; the concave inner surface lies in contact with the cornea. The rays emerge strongly divergent from this apparatus, and the illuminated fundus is seen with the naked eye.

BURNETT.

SIMI (417) made repeated sclerotomies at the equator of the ball in a case of glaucoma and in one of traumatic panophthalmitis. No disturbances attributable to the operation occurred, and the author therefore commends the study of it to his associates. The equatorial sclerotomy checked the glaucoma, where a deep corneal ulcer and peri-keratic swelling prevented him from performing an iridectomy or sclerotomy at the sclero-corneal margin. In the case of panophthalmitis the small operation immediately relieved the pain caused by the increased tension.

DANTONE.

DRANSART (419) tried suspension in cases of atrophy of the optic nerves without success. On the contrary, he obtained favorable results with this treatment in cases of neuro-retinitis hemorrhagica and in cases of miner's nystagmus.

MARCKWORT.

With the purpose of limiting enucleation to cases of intraocular tumors, DE WECKER (420) experimented on the cadaver in order to find whether it were possible to tear the optic nerve loose near the optic foramen in place of cutting it. He found the operation impossible. MARCKWORT.

BUCHNER (421) thoroughly investigated the emigration of leucocytes under chemical influences. Various vegetable and animal substances affect the leucocytes in different manners. He studied: (1) The irritation exerted on leucocytes by bacterial albumens (pneumo-bacillus protein), obtained by the digestion of bacteria masses with 0.5 per cent. potash lye. The substance was sealed in small glass tubes, sterilized, introduced under the skin, and the point broken ; it was found to be very active. (2) The irritation of products of decomposition. Glycocoll and leucin alone showed the slightest chemical action. (3) Vegetable caseine, gluten caseine, weakly alkaline, and legumin exerted a strong attraction on the leucocytes. Starch-flour acted negatively. (4) Transformation products of animal tissue. Albumen-peptone acted negatively, cement substance, on the contrary, actively. Muscle, liver, and kidney of the rabbit in 5- to 10-per-cent. solution showed a strong attraction. (5) Direct leucocytosis by chemical irritants. In the rabbit all substances which irritate the leucocytes, when injected in the aural veins, caused an increase in leucocytes. The ratio increased from 1:318 to 1:38. (6) The relation of the leucocytic attraction to inflammation and suppuration. This attraction of the leucocytes cannot be differentiated experimentally from the inflammatory irritation of the fixed tissue-

elements. Extensive experiments with the bacillus pyocyanus were undertaken in men.

III.—INSTRUMENTS AND REMEDIES.

422. Simi. On hot ocular douches. *Boll. d'ocul.*, vol. xii., p. 23.

423. De Wecker and Masselon. Sur les montures de lunettes. Un ophtalmo-statomètre. *Ann. d'ocul.*, vol. civ., p. 147.

424. Redmond, D. An electro-magnetic rotating needle for the discission of tough and calcareous secondary cataracts.

Simi (422) commends ocular douches with hot sublimate solutions (⅓ % and from 36–45° C.). He has had an irrigator constructed which keeps the solution at the temperature desired.

Dantone.

De Wecker and Masselon (423) speak of the necessity of ordering not only proper glasses, but also properly fitting frames. If the spectacles are to be made by an optician who cannot see the patient, besides the pupillary distance, the height of the bridge of the nose should be measured, the breadth of the cheeks, and the distance of the ears from the eyes. For the measurement of the bridge of the nose the authors take a base-line which passes through both pupils when the eyes are directed straight forward. The authors have had two particular instruments made for this purpose. Marckwort.

IV.—ANATOMY.

425. Antonelli. A contribution to the study of the morphological significance and the structure of the ciliary ganglion. *Giorn. dell' Assoc. dei naturalisti e med. di Napoli*, vol. i.

426. Nicati, W. La glande de l'humeur aqueuse, glande des procès ciliaires ou glande uvée. *Arch. d'opht.*, vol. x., No. 6, 1890.

Antonelli (425), on the ground of extensive comparative anatomical and histological researches, considers the ciliary ganglion as belonging to the oculomotor nerve, and the roots of the trigeminus and sympathetic as accessory. Dantone.

Nicati's (426) investigations treat of the apparatus secreting the aqueous humor in its histological, physiological, and pathological relations. He calls by the name "glande de l'humeur aqueuse" the portion of the uveal tract secreting this fluid, par-

ticularly the chorio-capillaris and the ciliary processes which form the secreting surface. He reviews the older and the newer ideas as to the seat of the secretion, nerve influence upon the intraocular tension, quantity, composition, and fluorescine coloring of the aqueous, and then treats of the histological relations of the "glande uvée" and its adnexa. He describes (1) an epithelium (outer or pigment layer, inner layer, or that composed of the cylindrical cells of the cilary processes); (2) fibres connecting the epithelium of the processes with the lens (in the eye of the rabbit, dog, and hen), representing the peculiar suspensory apparatus of the lens, independent of the posterior wall of the canal of Petit, which latter is formed of the hyaloid membrane with its connective-tissue reinforcements; (3) a capillary network—the chorio-capillaris anterior and posterior to the ora serrata, bounded inwardly by the lamina vitrea, outwardly by Sattler's membrane, and for this reason able to secrete fluid only anteriorly on the surface of the processes where the lamina vitrea ceases to be a continuous layer. The chorio-capillaris is a secretory organ and has nothing to do with the nutrition of the retina. (4) Arteries and veins (the ciliary vessels). (5) Muscle (smooth muscular fibres in the entire extent of the choroid and the ciliary muscle). (6) Nervous system with sensory motor and sympathetic fibres. (7) Orifices of the glands: the system of spaces of the suspensory ligament of the lens, the posterior chamber, and the communication between them and the spaces between the ciliary processes. (8) A receiving reservoir (the anterior chamber) and the excretory ducts: the iris and the spaces of Fontana, the crypts and stomata of the iris, and the lymph-vessels and perivenous lymph-spaces, which lead to the anterior and posterior ciliary veins.

V. MITTELSTÄDT.

V.—PHYSIOLOGY.

427. PIERINI. Discussion of some disputed points in physiological optics. *Giorn. della R. Accad. di Med. di Torino*, 11–12.

428. SEGAL, S. L. On the phosphenes which are seen at the point of pressure. Read before the Society of Physicians in Odessa. *Wratsch*, No. 48, 1890.

429. SCHIRMER, O. Does Weber's law hold good for the light sense? *Graefe's Archiv*, vol. xxxvi., 4, p. 121.

430. JERUSALEM, W. Laura Bridgman. The training of a blind deaf-mute. A physiological study. *Zeitschr. f. Psych. u. Phys. d. Sinnesorg.*, vol. i., part 6, p. 505.

431. RAEHLMANN. Physiologico-pyschological studies on the development of the faculty of recognizing faces in children, and in patients born blind who have been operated upon. *Zeitschr. f. Psych. u. Phys. d. Sinnesorganen*, vol. ii.

PIERINI (427) criticises, apparently justly, the views on the position of the centre of rotation of the eye, which Albertotti held in constructing his autoperimeter, and which Landolt held in the investigations with his chiastometer. Secondi's opinion, that the angle α changes in different positions of the visual lines while the accommodation remains the same, is also considered untenable by the author. DANTONE.

Besides the well-known pressure phosphene opposite the point of pressure, SEGAL (428) has observed a second less intense phosphene in the direction of the point of pressure, and especially marked when the pressure is intermittent. The author explains this as caused by the conduction of the pressure through the vitreous to the retina opposite. Segal saw the phosphene directly before him when intermittent pressure was made on the cornea. HIRSCHMANN.

SCHIRMER (429) sought to establish the truth of Weber's law for the light sense by a careful study of adaptation within certain limits of illumination. The experiments of Aubert and Helmholtz, which apparently disproved the law, were criticised, and by a series of original experiments the author attempted to prove :

(1) That Weber's law of the least perceptible differences holds good for the light sense within the limits of illumination 1-1000 M K, if the eye is permitted to exercise its complete power of adaptation ; the truth of the law is therefore dependent upon certain physiological conditions.

(2) The truth of Weber's law can be explained only by means of the physiological process of adaptation.

(3) In the normal eye the adaptation does not, or at least not always, proceed regularly in the dusk when the daylight decreases.

Sections VI.–XI. Reviewed by DR. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

430. WEISS, L. Contributions to the anatomy of the orbit. iii.—On the relation of the orbit to the various forms of the head and face. Tübingen, 1890. H. Laupp.

431. BERRY, G. On myopia. *Ophth. Rev.*, vol. ix., p. 327.

431a. P. SMITH. A reply to the preceding. *Ibid.*, p. 345.

432. KROTOSCHIN. Anatomical contributions regarding the development of myopia. *Arch. f. Augenheilk.*, vol. xxii., p. 393.

433. JONG, W. de. A contribution to the development of myopia. Examination of the eyes of 3,930 school children in Leyden. *Zeitschr. f. Schulgesundheitspflege*, 1890, No. 12, p. 739.

434. KOTELMANN. Dr. Francis Warner's report on the examination of fourteen London schools. *Ibid.*, No. 12, p. 697.

435. GALEZOWSKI. A study of some grave varieties of myopia and the means of curing them. *Rec. d'opht.*, 1890, p. 510 and 571.

436. MARTIN, G. Astigmatic amblyopia. *Ann. d'ocul.*, vol. civ., p. 101.

437. KOLLER, C. On the determination of astigmatism with the ophthalmometer (Javal-Schiötz). *Jour. Amer. Med. Assoc.*, Sept. 13, 1890.

438. GALEZOWSKI. On the diagnosis of irregular astigmatism and its correction by means of cylindrico-conical lenses.

The view is held in many quarters that the conformation of the orbit has an influence on the form of the eyeball, and consequently on its refractive condition. WEISS (430), in order to test this relation, reports accurate measurements made on more than one hundred skulls.

BERRY (431) states that benign myopia differs ætiologically and anatomically from myopia with affection of the choroid. The worst cases of the latter category are found in the uneducated classes, and are more frequently seen in the clinic than in private practice. It is not proven that convergence increases the sagittal diameter of the ball, and there are grounds for opposing this view. WERNER.

P. SMITH (431a) replies by giving statistics of 4,000 cases of myopia. In 2,000 private cases 64 had myopia over 10°. While in 2,000 hospital patients only 27 had myopia over 10° (7 of these had 20° or more, while 8 of the former only reached this; this is in favor of Berry's contention). There is no definable boundary between benign and malignant myopia, and it is difficult to prove it. In a hypermetropic eye the convergence does not cause the same strain on the sclera (as shown by Emmert). In

unilateral myopia the effort of convergence is made, but is impeded by the shape of the eyeball. WERNER.

KROTOSCHIN (432), after making a great number of measurements, accepts Stilling's theory, that a low orbit disposes to myopia, and a high one to emmetropia or hyperopia.

GALEZOWSKI (435) speaks of myopia in general. According to him, myopia almost always remains stationary when accompanied with astigmatism. Myopia sometimes develops at the age of 2–3 years. Even in low degrees of myopia glasses should be worn for near work. MARCKWORT.

MARTIN (436) publishes another long paper, in which he reports much new clinical matter, which serves to support his idea of astigmatic amblyopia. By astigmatic amblyopia the author understands an amblyopia which, as a result of astigmatism, develops in one meridian. A tabular report is given of the condition of 108 eyes with astigmatic amblyopia. When a considerable astigmatism develops from a trauma in that period of life before the retina has completely gained its functions, no astigmatic amblyopia occurs. When an astigmatic accommodation corrects a corneal astigmatism, no amblyopia develops. There is not so great a tendency to this correction when the other eye is not astigmatic and has good vision; therefore the amblyopia is usually more marked in cases of monocular astigmatism than in cases of binocular. The relation of objective to subjective astigmatism is also discussed and illustrated with statistics. In 108 cases, the objective and subjective astigmatism were equal in 49. In myopes the subjective astigmatism is often greater than the objective, while the contrary relation is found in simple hyperopic astigmatism. Martin finds the different acuteness of vision at the different axes, and designates one the maximum acuteness and the other the minimum acuteness of vision. The acuteness of vision usually found corresponds nearer to the maximal meridian acuteness than to the minimum meridian acuteness. MARCKWORT.

GALEZOWSKI (438) has had cylindrico-conical lenses made to correct irregular astigmatism, and especially irregular lenticular astigmatism. While ordinary cylindrical lenses are segments of the surface of a cylinder, the new lenses represent portions of the surface of a cone taken parallel to its axis, and so having a different curvature at different distances from the apex of the cone. MARCKWORT.

VII.—LIDS.

439. FRIEDENWALD, H. Cases of phlegmonous gangrene of the lid. *Amer. Jour. of Ophth.*, vol. vii., p. 285.

440. HALLOPEAU. Hydro-adenoma of the lid, complicated with epithelioma. *Union méd.*, 1891, No. 4.

441. AMORIN. On the restoration of the lids by skin-grafting. *Thèse de Paris*, 1890.

442. FONTAN. Blepharoplasty by scissor-shaped flaps for the total restoration of both lids. *Soc. d'opht.*, Nov. 4, 1890.

443. RAY, J. M. Report of a case of transplantation without a pedicle for cicatricial ectropion. *Jour. Amer. Med. Assoc.*, Oct. 11, 1890.

444. ROBERTSON, A. Note on the treatment of severe senile entropium. *Edinburgh. Med. Jour.*, 1890, No. 1526, p. 497.

445. HARLAN, G. C. New operation for symblepharon. *Ophth. Rev.*, vol. ix., p. 351.

446. SNELLEN, H. New method of treating symblepharon. *Trans. Ophth. Soc.*, vol. x. p. 207.

447. SCHEFFELS, OTTO. On advancement of the levator tendon. *Graefe's Archiv*, vol. xxxvi., 4, p. 374.

448. BROWNING, F. W. Associated contraction of the levatores palpebrarum superiorum with the internal recti. *Trans. Ophth. Soc.*, vol. x., p. 187.

FRIEDENWALD'S (439) case was that of a little girl who scratched herself on the temple. In the course of a few hours great swelling set in, followed by a gangrene and sloughing of almost the entire upper lid. The ciliary margin, however, was not involved. Eyeball not affected. Treatment constitutional, and local application of iodoform. BURNETT.

FONTAN (442) made a plastic operation in a patient, the skin of whose face had been extensively destroyed by carcinoma. There were wanting both left eyelids, the outer half of the left eyebrow, the outer portion of the forehead, the temple, and the cheek on the left side. A forehead flap was taken for the lower lid, and a nasal flap for the upper. Since the flaps crossed each other, like a pair of scissors, he called the operation blepharoplasty with scissor-shaped flaps. MARCKWORT.

RAY'S (443) case was an ectropion of the upper lid, and the raw surface left by its detachment was 2 by 1¼ inches. This was

covered by integument taken from the arm, according to Wolfe's method, and kept in place by gold-beaters' skin and non-contractile collodion. Union was perfect. Three months after, the flap had shrunken to 1 by ¾ inch, but the lid covered the cornea nicely. BURNETT.

In symblepharon of the lower lid, HARLAN (445) dissects up the adhesion and turns in a flap of skin through an incision in the lid; the flap having its raw surface in contact with the inner surface of the lid, and its sound surface toward the eyeball. WERNER.

SNELLEN (446), in bad cases of symblepharon, passes a pediculated skin flap in through a button-hole in the lid, the skin being in contact with the eye. His object in ptosis is to shorten the levator tendon by sutures, without cutting the skin. He recommends subconjunctival injection of sublimate 1:5000 for episcleritis. WERNER.

For entropium and trichiasis of the upper lid, SCHEFFELS (447) recommends the advancement of the levator tendon. This produces a secure cicatricial attachment between the fibres of the orbicularis on one side and the upper tarsal margin, with the fascia and tendon of the levator on the other, while at the same time advancing the tendon and changing the direction of its point of attachment.

In BROWNING'S (448) patient, with the action of the internarectus an elevation of the upper lid of the same side was observed, with convergence a similar elevation of both upper lids, and on looking downward the lids reached only to the horizontal meridian. WERNER.

VIII.—LACHRYMAL APPARATUS.

449. CAUDRON. Two cases of acute dacryo-adenitis. *Soc. d'Opht. de Paris*, Nov. 7, 1890.

450. SIMI. A case of abscess of the sac cured with anilin oil. *Boll. d'ocul.*, vol. xii., 24.

CAUDRON (449) reports two new cases of inflammation of the lachrymal gland. In the first case the swelling of the gland recurred at the menstrual periods, becoming each time more severe. The patient was seen but once. In the second case Meyer made a puncture through the conjunctiva and freed a clear fluid with good results. MARCKWORT.

SIMI (450) reports another case of abscess of the sac in which anilin oil (8 drops to 100 gr. water) acted as a valuable antiseptic.

DANTONE.

IX.—MUSCLES AND NERVES.

451. BERRY, E. Note on the immediate effect of tenotomy on the concomitancy of a squint. *Trans. Ophth. Soc.*, vol. x., p. 202.

452. MCACHRAN. Laceration of the internal rectus. *Amer. Jour. of Ophth.*, vol. vii., 11, p. 364.

453. LANDOLT, E. On muscular asthenopia. *Arch. d'opht.*, 1890, No. 6, p. 509.

454. ROOSA, ST. J. Muscular asthenopia. *Ophth. Rev.*, vol. ix., p. 282.

455. WEBSTER, D. A case of paralysis of the superior oblique cured by tenotomy of the superior rectus of the same side. *Med. Rec.*, Jan. 7, 1890.

456. RAYMOND, P. A case of nuclear ophthalmoplegia externa. *Gaz. des hôp.*, 1890, No. 126, p. 1167.

457. MOREL, F. A contribution to the study of ophthalmoplegia externa. *Thèse de Paris*, 1890.

458. BOUVERET et CURTILLET. Bilateral ophthalmoplegia externa of embolic origin. *Gaz. hebd.*, 1890, No. 40, p. 512.

459. DEHOVE. Bilateral paralysis of the third nerve, of hysterotraumatic origin. *Sem. méd.*, 1890, p. 455.

460. GUENDE. The muscular paralyses of the eye. *Rec. d'opht.*, 1890, No. 12, p. 705.

461. ROMANO–CATANIA. On a form of paralysis of the ocular muscles accompanied by myopia.

462. CHRIST. On the ætiology of nuclear ophthalmoplegia. *Deutsches Arch. f. klin. Med.*, vol. xlvi., p. 496.

463. LANDOLT, E. Ocular torticollis. *Bull. méd.*, 1890, p. 573.

464. EVERSBUSCH, O. A case of traumatic ciliary neuralgia cured by the galvano-cautery. *Münchener med. Wochenschr.*, 1890, No. 51.

465. ALLPORT, F. Some remarks on the relief of remote neuroses by the restoration of ocular equilibrium. *Amer. Jour. of Ophth.*, vol. vii., 11, p. 367.

BERRY (451) measures the convergence not alone when fixing in the median line, but also about 30° to either side. The results

will only be the same when the concomitancy of the eyes is complete. He finds that the squint remaining after a tenotomy increases in amount on fixing toward the side of the squinting eye.

WERNER.

LANDOLT (453) objects to the opinion expressed at the Berlin Medical Congress that muscular asthenopia does not exist. He gives a brief description of the symptoms, and reports some observations respecting treatment which go to show, as he has previously stated, the advantage which advancement has over tenotomy.

V. MITTELSTÄDT.

ROOSA (454) is of the opinion that insufficiency of the ocular muscles is usually a consequence of an error of refraction; his reasons for coming to this conclusion being that he had always found prisms unsatisfactory, and that the correction of astigmatism removed the symptoms and the necessity for prisms. Tests for insufficiency, etc., are delusive owing to the varying strength of the muscles, and equilibrium exists only in a moderate percentage of people who are not asthenopic. The author admits the existence of *neurotic* asthenopia, which may not be relieved by correction of refractive errors, but in these cases tenotomies and prisms are only means of treating these cases by suggestion.

WERNER.

The paralysis of the superior oblique in WEBSTER'S (455) case was syphilitic, but did not yield to active anti-syphilitic treatment. A tenotomy of the superior rectus resulted in an over-correction, which was remedied later by an advancement of the same muscle. The cure was complete, there being no diplopia. BURNETT.

ROMANO-CATANIA (461) reports a case of congenital bilateral paralysis of the muscles supplied by the third and sixth nerves, excepting the sphincter pupillæ and the muscle of accommodation, in a man who was excessively myopic. Since this was apparently due to a functional disturbance of the nuclei of origin of the nerves in question, which had developed in fœtal life, the author, on account of the non-disturbance of innervation of the interior muscles of the eye, endeavors to support the opinion of many others (Hensen and Völkers, Kahler and Pick), according to whom the nucleus of origin of the third nerve has an extent of some 10 *mm*, through which are distributed the individual nuclei of the branches supplying particular muscles. In the case in question the middle and posterior portions of the nuclei are those at fault, since the nuclei for the interior muscles of the eye lie in the anterior portion. The symmetrical limitation of the disturb-

ance may be anatomically explained by the supposition that the nucleus is supplied by several small arteries which do not anastomose (terminal arteries); a disturbance in the circulation of one or more of these terminal arteries would therefore cause a disturbance in the particular portion of the nucleus supplied. Similar cases have been described by Schroeder and Rählmann. The extreme myopia in this case was explained by the author as being due to the pressure exerted by the superior oblique, which muscle alone preserved its function, and which plays an important rôle in the development of myopia in general and especially when the vertical diameter of the orbit is short. DANTONE.

CHRIST (462) showed a complete paralysis of the fourth nerve and an incomplete paralysis of the third in both sides. At the autopsy a gliosarcoma was found involving the pineal gland, the region of the corpora quadrigemina, and the posterior portion of the third ventricle.

LANDOLT (463) described two cases of torticollis after paralysis of the fourth nerve. In order to correct the disturbing double vision the patient had brought his head into this peculiar position. The head was inclined toward the shoulder and the face turned to the opposite side. Tenotomy was advised but refused.

MARCKWORT.

EVERSBUSCH (464) reported a case of injury to the eye with a flax comb. The vision became normal, but there was pain in accommodation. A small cicatrix was seen at the corneo-scleral margin. When this was destroyed with the galvano-cautery, all difficulty was relieved.

ALLPORT (465) believes that properly performed tenotomies of the external muscles will in many cases relieve headaches, but does not think that they can affect remote neuroses, such as chorea, epilepsy, etc. BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

466. SNELL, S. Acute cellulitis of orbit; fatal result. *Trans. Ophth. Soc.*, vol. x., p. 51.

467. DRANSART. Symptomatologie, diagnostique, étiologie et traitement des hydrarthroses orbito-oculaires séreuses. *Jour. d'ocul. du Nord de la France*, 1890, No. 1, p. 6.

468. SPENCER WATSON. A case of osteoma of the infra-superciliary region, with the specimens removed by operation. *Trans. Ophth. Soc.*, vol. xi., p. 44.

469. Rockliffe, W. C. Case of cephalic tetanus following penetrating wound of the orbit; recovery. *Trans. Ophth. Soc.*, vol. x., p. 46.

Snell's (466) patient, a girl of 14, developed cellulitis in the right orbit as the result of diseased molar teeth which had caused slight necrosis of the jaw. She died of coma in spite of the local improvement which resulted from treatment. Werner.

Dransart (467) believes that a serous effusion may collect beneath Tenon's capsule, and, by pressure on the optic nerve, lead to its atrophy. This exudation may be allowed to escape by means of an operation suggested by Dransart. Capsulotomy is also indicated in cases of phlegmonous tenonitis. In various other diseases Dransart makes a capsulectomy. Marckwort.

Watson (468) removed a small ivory osteoma from a woman æt. 30. It lay quite free in the subcutaneous tissue of the right eyebrow. Werner.

In Rockliffe's (469) case, a boy æt. 7, injury to the left eyebrow was followed by ptosis and facial paralysis, accompanied by trismus and slight pharyngeal and respiratory spasms. Recovery took place in fourteen days. Werner.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

470. Andrews, J. A. Purulent ophthalmia; the ætiology and treatment of the disease. *New York Med. Jour.*, vol. lii., No. 25, p. 681.

471. Schmidt, E. Report of the eye department of the Marine Hospital in Sebastopol for the year 1889. *Med. supplement to the Moskoi Sbornik*, Oct., 1890.

472. Darier. The surgical treatment of granular conjunctivitis. *Soc. d'Opht. de Paris*, Dec. 2, 1890.

473. Reid, T. Lymphoma of the conjunctiva. *Trans. Ophth. Soc.*, vol. x., p. 57.

474. Morton, A. S. Fibro-myxoma of the plica semilunaris and lower conjunctival sac. *Ibid.*, p. 64.

475. Lopez, Enrique. A clinical note on ophthalmia neonatorum. *Revista de Ciencias Medicas*, Dec. 5, 1890.

Andrews (470) found the gonococcus 17 times in 17 cases of acute gonorrhœa, 108 times in 144 cases of chronic gonorrhœa, and in every case of 72 instances of purulent ophthalmia in adults

and in 122 of the newly-born. He thinks there is often a purulent ophthalmia in children under three weeks which is not specific. His main reliance in treatment is in an instillation of a 2-per-cent. solution of nitrate of silver, once or oftener a day, according to indications; the eye to be kept clean with frequent washings of a boric-acid solution, and cold to be constantly applied. In a total of 153 cases treated, there was a total loss of 1 eye in 8 cases.

BURNETT.

According to SCHMIDT'S (471) report, the chief contingent of eye diseases is formed of cases of trachoma, in which he includes the so-called folliculosis and the catarrhus follicularis. He has often observed the development of undoubted trachoma from the lighter forms of folliculosis. In its early stages, trachoma yields readily to treatment. After being cured, the patient should still be kept under observation for a week or two, to guard against relapses. The formation of follicles on the tarsal surface is always a sign of the long duration of the disease, and demands a more extended treatment. In the first stages of trachoma the follicles are present only in the cul-de-sac, and may easily be overlooked. The only cause is infection. The treatment consists in the early squeezing out of the follicles, which usually prevents extensive cicatrization; and later in the use of 1-per-cent. to 2-per cent. nitrate of silver, 2 per cent. acetate of lead, or the stick of sulphate of copper, which prevents the involvement of the cornea.

HIRSCHMANN.

DARIER (472) operates on the severer cases of granular conjunctivitis after Sattler's manner. He, however, lays less weight on the scraping than on the brushing and washing of the cul-de-sac with sublimate solution 1 : 500.

MARCKWORT.

REID (473) contributes an interesting microscopical study of conjunctival inflammations, illustrated with microphotographs. Lymphoid infiltration is shown to be a frequent condition in various forms of conjunctivitis. In recent follicular conjunctivitis, the follicles have a finely reticulated stroma, are not surrounded with any definite capsule, and probably increase in size by the addition of surrounding lymphoid cells; as the follicle develops the connective tissue in the centre increases, and fatty or other degeneration occurs. Probably the formation of follicles is secondary to the general lymphoid infiltration. A follicle may burst and discharge on the surface. In cases of long standing goblet cells become numerous, and some may become involuted

and thus be the beginning of a mucous follicle forming a vesicle in the retrotarsal fold. The fibro-cellular infiltration is not limited to genuine trachoma, but may occur in chronic follicular cases, so that the distinction between these two diseases is merely clinical. WERNER.

MORTON (474) gives an illustration of two small fibro-myxomatous tumors *in situ;* they were firm and pinkish in color, and were growing for three or four months. Patient aged 37. WERNER.

LOPEZ (475), after a twin delivery, found purulent ophthalmia on the third day in the last-born infant, which was delivered with the forceps, while the first-born infant, which was delivered easily, did not contract the disease. This proves (1) that children born of a leucorrhœic mother do not necessarily get purulent ophthalmia; and (2) that the infection takes place through the direct contact of the secretion with the conjunctiva, for the case could be explained on no other grounds.

476. FEILCHENFELD. The therapy of superficial corneal infiltration. *Zehender's klin. Monatsbl. f. Augenheilk.*, vol. xxviii., p. 502.

477. BOULLET. Étude comparative du traitement des abscès de la cornée par la chaleur et la froid. *Thèse de Lyon*, 1890.

478. RANDOLPH, R. S. Mild solutions of corrosive sublimate in the treatment of keratitis. *Johns Hopkins Hosp. Bull.*, vol. i., 4, p. 46.

479. CANT, W. E. Case of keratitis from paralysis of left fifth nerve; injury to fifth nerve within cranium. *Trans. Ophth. Soc.*, vol. x., p. 233.

480. DESPAGNET. Traitement du keratocone pellucide. *Soc. d'Opht. de Paris*, Nov. 4, 1890.

FEILCHENFELD (476) scratches out superficial corneal ulcers with a gouge or needle, and then applies an antiseptic dressing. The results are good.

RANDOLPH (478) has found instillation of solution of the mercuric chloride, from $\frac{1}{6000}$ to $\frac{1}{12000}$, every four hours useful in the treatment of all forms of keratitis. Atropine solution is used in addition. BURNETT.

CANT's (479) patient had complete paralysis of all motor and sensory branches of the fifth nerve. The boy was aged sixteen, and had received a blow from a reaping-hook two or three weeks

before, the point of the hook having penetrated the squamous portion of the temporal bone to the depth of an inch. Severe keratitis set in, which improved after slight return of sensibility.

WERNER.

DESPAGNET (480) reported three cases of keratoconus treated in different manners. In the first case a small ulcer formed at the apex of the cone. A compressed bandage was used and later, after cauterization with nitrate of silver, the cone became flatter, but a slight keratoglobus resulted. In the second case, one eye was treated with eserine and a compress bandage, while the other eye was not treated. The conus increased equally in both eyes and the vision decreased correspondingly. In the third case he excised a crescentic bit from the entire thickness of the cornea after Galezowski's method. The wound cicatrized in a month, but six months later became infiltrated and an ulcer developed. This was cauterized with the galvano-cautery, a paracentesis was made and a compress bandage applied, and the ulcer healed. Despagnet, therefore, recommends the treatment by excision of a crescentic bit of cornea. In the discussion de Wecker advised against operative treatment. Of all the operations suggested that of v. Graefe is still the best. Meyer operates according to v. Graefe. Abadie perforates the cornea with the galvano-cautery. Jocqs favors eserine and a compress bandage. Gillet de Grandmont saw a good result after v. Graefe's operation, but will not hesitate to open the anterior chamber in the excision. Parinaud scarifies the cornea. MARCKWORT.

481. MILLIKEN, B. T. A case of foreign body remaining in the eye for twenty years, followed by abscess in the scleral wall. *Med. News*, Nov. 8, 1890.

MILLIKEN (481) relates an interesting case of a piece of cap remaining in the eye, with fair vision for twenty years. It then gave rise to disturbance which was repeated at intervals for ten more years. The foreign body had entered at the edge of the cornea and passed through the iris and lens, which latter was circumscribedly opaque. With the ophthalmoscope the body could be seen projecting into the vitreous. It later formed a projection on the outer surface of the sclera and finally M. punctured the circumscribed tumor and let out three or four drops of pus and some metallic detritus. The patient has now V = $\frac{3}{30}$ and all symptoms of irritation have disappeared. BURNETT.

Sections XII.–XXI. Reviewed by Dr. P. SILEX.

XII.—IRIS.

482. Brailey. Double microphthalmos with defective development of iris, teeth, and anus. Glaucoma at an early age. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 139.

483. Hill, Griffith. Case of tuberculosis of the iris. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 84.

Brailey's (483) patient, a girl æt. 18, had only a crescentic portion of iris left at inner side in the right eye, and three small, isolated bits in the left eye. The corneæ measured 9 *mm* in diameter. Myopia = 12 D. Vision only = counting fingers. Disks cupped. Werner.

In Griffith's (483) patient, a child æt. 7 months, a yellowish non-vascular tumor grew at ciliary border of the iris at outer side, pupil closed by delicate membrane and there was slight bulging of sclero-corneal region. No history or signs of phthisis or syphilis. On account of further formation of nodules the eye was enucleated. The child died nine months after from tubercular meningitis. The growth exhibited a tubercular structure, but bacilli could not be demonstrated probably owing to hardening fluid having been bichromate of potash. The paper concludes with an interesting review of previously recorded cases and a list of references.

Werner.

XIII.—CHOROID.

484. Brailey. Cyclitis, probably of gouty origin. *Trans. Soc. of United Kingd.*, vol. x., p. 96.

Brailey's (484) patient, a man æt. 36, after having had six attacks of gout in the feet and limbs, became suddenly affected with well-marked cyclitis in one eye which relapsed three times. The eye was acutely tender to the touch and periphery of anterior chamber deep. Werner.

XIV.—GLAUCOMA.

485. Collins, J. T. Glaucoma after extraction of cataract. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 108.

486. Morton, Stanford. Hemorrhages on the disk in a case of glaucoma (with plate). *Ibid.*, vol. x., p. 153.

487. Smith, Priestley. On the size of the cornea in relation to age, sex, refraction, and primary glaucoma. *Ibid.*, vol. x., p. 68.

488. KEYSER, P. A case of glaucoma fulminans of peculiar interest. *Am. Jour. of Ophth.*, No. 8, 1890.

In this interesting paper COLLINS (485) describes the history and pathological condition of 10 eyes which were lost from glaucoma after cataract operations. In 9 cases the cataract was senile, in one probably traumatic. In 5, one eye was operated on without any subsequent symptoms of glaucoma, in 3 the increased tension came on after needling, and in 3 others it was accompanied by iritis with keratitis punctata, and sympathetic ophthalmia occurred.

The glaucoma came on in from 3 months to nearly 2 years after the operation. Iridectomy was performed in 9 cases of extraction. Pathological examination revealed adhesion of lens capsule to the wound except in one instance where the hyaloid of the vitreous was adherent to it, and in this case the lens had been removed in its capsule, and in all cases except this some cortical remains had been left in the peripheral portion of the capsule. The angle of the anterior chamber in the coloboma was blocked in all cases, and elsewhere there was peripheral adhesion of the root of the iris except in the kerato-iritic cases where there was cellular infiltration of lig., pectinatum iridis, etc.

From the beginning of 1885 to the middle of 1889 1,405 senile cataracts were extracted at the Moorfields Hospital and 9 of these were lost from glaucoma, or 0.64 per cent. WERNER.

In MORTON'S (486) case the hemorrhages on the margins of the cupped disk were associated with aneurismal dilatations of some vessels. WERNER.

P. SMITH'S (487) investigations were undertaken to test the truth of a previous suggestion of his, that small corneas were associated with a liability to primary glaucoma. 1,000 normal eyes were examined; ages of subjects 5 to 20. Average horizontal diameter 11.6 *mm*, the maximum being 13.5 *mm*, and the smallest 10.5 *mm*. The cornea attains its diameter very early in life—about the age of 5. The relation which sex and refraction have to the diameter of the cornea is almost negative. 178 glaucomatous eyes examined gave an average diameter of 11.12 *mm*, maximum 12.5, minimum 10 *mm*. Corneas with less than 11 *mm* diameter formed 25 per cent. of glaucomatous and less than 4 per cent. of normal eyes. The smallness of the cornea is not a consequence of the glaucoma, as patients who had one eye normal had the same-sized corneas in both eyes. Two questions still remain

to be positively determined—namely, are small, corneas associated with a proportionally small lens and eyeball. WERNER.

In KEYSER'S (488) case after an extraction with a large iridectomy there remained a thickened capsule which was finally drawn out through a small corneal incision by means of a Tyrrel hook. After this latter operation there developed in a few hours an attack of glaucoma fulminans, which was subdued by active treatment, but recurred twice subsequently. The final result however, was good. BURNETT.

XV.—SYMPATHETIC OPHTHALMIA.

489. GALEZOWSKI. Du mode de transmission de l'ophtalmie sympathique et de son traitement. *Soc. d'Opht. de Paris*, Oct. 7, 1890.

490. GOODE, G. A. A case of sympathetic ophthalmia two weeks after enucleation of the injured eye. *Jour. Am. Med. Asso.*, July, 1890.

491. MEYER. Quelques remarques sur l'ophtalmie sympathique. *Soc. d'Opht. de Paris*, Nov. 4, and *Rev. génér. d'opht.*, 1890, No. 11., p. 481.

492. DE WECKER. Resection simple du nerf optique *Soc. d'Opht. de Paris*, Nov. 4, 1890.

493. ROLLAND. Traitement préventif de l'ophtalmie sympathique. 1890, p. 527.

GALEZOWSKI (489) assumes that sympathetic inflammation is conveyed from one eye to the other through the ciliary nerves and their lymph channels, and not through the optic nerves. He bases his opinion on the anatomical investigations of Poncet in opposition to Knies, Leber, and Deutschmann. MARCKWORT.

GOODE'S (490) patient was a boy of eleven years whose cornea was ruptured by a blow from a stone. Two weeks after the eye was enucleated, and fourteen days later a sero-plastic iritis set up in the fellow-eye. Vision at last report only $\frac{2}{60}$. BURNETT.

MEYER (491) in a long paper discusses Deutschmann's experiments on ophthalmia migrans in their relation to clinical observations. Meyer grants the value of these investigations in part, but believes that sympathetic ophthalmia in man does not arise from the simple migration of bacteria to the second eye through the optic nerve. He advises early enucleation. In glaucoma absolutum Meyer has made æquatorial punctures for several years.

MARCKWORT.

DE WECKER (492) advises resection of the optic nerve in place of enucleation for sympathetic ophthalmia. His operation differs from those of Schweigger and H. Pagenstecher, in that he endeavors to limit it as much as possible to the nerve itself. He draws the eyeball forward with his "crochet articulé," and seizes the tense nerve with his "ciseaux compresseurs," and excises as large a piece of the nerve as possible. MARCKWORT.

ROLLAND (493) believes enucleation to be the only safe and commendable operation for sympathetic trouble. He warns practising physicians not to be misled by de Wecker's paper into the belief that enucleation can be substituted by de Wecker's æquatorial punctures. de Wecker's paper is severely criticised.

MARCKWORT.

XVI.—LENS.

494. BERRY. A case of spontaneous purulent hyalitis occurring nine months after a successful cataract extraction, with remarks. *Trans. Ophth. Soc. of the United Kingd.*, vol. x., p. 160.

495. COLLINS, E. T. Lens with epithelium on its posterior capsule and other peculiar structural changes. *Ibid.*, vol. x., p. 165.

496. TYNER, J. D. Preliminary capsulotomy in the extraction of cataract. *New York Med. Jour.*, Sept. 30, 1890.

497. LIPPINCOTT. On intra-ocular syringing in cataract extraction, with a report of fifty-three operations. *Amer. Jour. of Ophth.*, July, 1890.

498. MICHEL, M. History of the admission of light in the after-treatment of cataract operations. *Med. News*, Sept. 20, 1890.

499. SNELL, S. On artificial ripening of cataracts with special reference to Förster's operation of trituration. *Trans. Ophth. Soc. of the United Kingd.*, vol. x., p. 214.

500. THOMPSON, J. T. Note on a case of hereditary tendency to cataract in early childhood. *Ibid.*, vol. x., p. 141.

501. WEBSTER, D. Traumatic dislocation of the crystalline lens with increased tension and severe pain; relief of pain and restoration of useful vision follow extraction. *New York Med. Jour.*, Sept. 13, 1890.

In BERRY'S (494) case the patient, a glass-blower, returned to his occupation a month after a successful cataract extraction; nine

months later he presented himself with a purulent hyalitis, with hypopyon. The eye was enucleated. No adhesion of iris to the wound, which was firmly united. The suppuration seemed to have begun just behind the corneal margin. A second similar case is recorded in the paper. The patient was a cook. WERNER.

COLLINS' (495) patient lost the eye from secondary glaucoma due to a choroidal growth. The lens exhibited a single layer of nucleated epithelial cells lining the posterior capsule, and a central band of hyaline bodies. WERNER.

TYNER (496), after dilating the pupil, introduces a Bowman needle and ruptures the lens capsule in its upper quadrant. The incision is then made and the extraction performed in the customary manner. The aqueous does not escape. BURNETT.

LIPPINCOTT (497) tabulates fifty-three extractions, in all of which a preliminary iridectomy had been made in which he washed out the corticalis with a boric-acid solution by means of a specially devised syringe. His successes were ninety-eight per cent.; failures, two per cent. BURNETT.

MICHEL (498) shows that the reaction against dark rooms and heavy bandages began as far back as 1801 with Beer, and that Walton and others for closing the lids used adhesive straps only, in 1870. Among Americans, Agnew of New York and Lewis and Roberts of Philadelphia used the same methods. The method was brought more prominently forward however by Dr. Chas. E. Michel of St. Louis, and has since been followed particularly by Chisolm of Baltimore. BURNETT.

SNELL (499) speaks favorably of Förster's operation. He reports nine cases, only two of which were unsuccessful, and one of these had no cortical capacity. In no case was there iritis or any complication. In the discussion Tweedy stated that he had frequently operated on unripe cataracts with successful result, which he attributed to the peripheral rupture of the capsule. Hill-Griffith had tried trituration in twenty-eight cases on the anterior portion of the capsule, and once a partial dislocation of the lens occurred. Eales had seen no bad result in thirty cases. In a number of cases there was slight adhesion between the iris and the anterior capsule. Vitreous was never lost. Brailey does not favor the extraction of unripe cataracts. WERNER.

THOMPSON (500) gives a genealogical tree of five generations in which eleven persons developed cataract before five years of age; in two generations this seems to have been transmitted through

the male line. The cataracts were not zonular but more like senile in density. WERNER.

WEBSTER'S (501) patient was a man of sixty years. In extracting the lens he did not use the trident devised by Dr. Agnew, but after making a large incision introduced a wire spoon and picked the transparent lens out in its capsule. BURNETT.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

502. EDMUNDS, W. Sections of retina from a case of leucocythæmia. *Trans. Ophth. Soc. United Kingd.*, vol. x., p. 157.

503. EPÉRON. Sur quelques symptomes tabiformes de l'amblyopie toxique. *Rev. méd. de la Suisse romande*, Aug. 8, 1890.

504. EWETZKY. On the pathological anatomy of retinitis pigmentosa. *Wjestnik Ophth.*, No. 6, 1890.

505. GARAFOLO. A case of quinine amaurosis. *Wiener med. Blätter*, No. 15, 1890.

506. GALLEMAERTS. Amblyopia from sulphuret of carbon. *Ann. d'ocul.*, vol. civ., p. 154.

507. GRAND, CLÉMENT. Efficacité des injections d'antipyrine dans trois cas d'héméralopie symptomatique de rétinite pigmentaire. *Ann. d'ocul.*, vol. civ., p. 232.

508. HIRSCHBERG. On retinal changes due to old age. *Centralbl. f. prakt. Augenheilk.*, vol. xiv., p. 322.

509. KOENIG. Les thromboses artérielles de la rétine. *Rec. d'opht.*, 1890, p. 697.

510. LAWFORD, B. Embolism of a branch of the arteria centralis retinæ. Right eye. *Trans. Ophth. Soc. United Kingd.*, vol. x., p. 153.

511. WERNER. Abnormal retinal veins. *Ibid.*, vol. x., p. 151.

512. FERNANDEZ. Treatment of detachment of the retina by scleral puncture. *Anales de la Real Academia de Ciènces med. fis. y natur*, Dec., 1890.

In EDMUNDS' (502) case, in addition to hemorrhages, the retina showed thickening of the walls of the arteries, and clots of white corpuscles in the veins. WERNER.

EPÉRON (503), in cases of intoxication amblyopia, has frequently seen tabic symptoms, such as the Argyll-Robertson pupil, myosis, loss of knee-jerk, Westphal's symptom, etc. The chief differential points are the preservation of the periphery of the visual field and the existence of a central scotoma. MARCKWORT.

In examining the eyes of a man of forty-four, dead of delirium tremens, who had been blind from early youth, EWETZKY (504) found the retinas moderately pigmented, particularly between the equator and the disk.

Microscopically the inner layers of the retina were seen to be well preserved. The outer molecular layer, the outer granular layer, and the rods and cones were entirely wanting. They were replaced by a thin layer of reticular connective tissue lying on the inner granular layer. In the nerve-fibre layer the nerve fibres were thickened at many points, as in retinitis albuminurica. The vessel walls were thickened, the lumen narrowed and at some points obliterated, and many vessels were transformed into connective tissue. Pigment in the form of free-lying granules was present in all the layers of the retina, but particularly in the nerve-fibre layer, and less chiefly about the vessels. The pigment epithelium was altered, the form of the cell remaining usually normal, but the pigment altogether wanting or much decreased, and often accumulated between the cells.

The author concludes from his studies that retinitis pigmentosa is a disease of the retinal vessels, which leads to atrophy of the outer layers of the retina and secondary hypertrophy of the connective tissue. The pigment is washed out of the atrophic pigment epithelium by the fluid currents in the inner layers of the retina. HIRSCHMANN.

GARAFOLO'S (505) patient, a man, æt. sixty-one, took 15 grm. of quinine. Six days later R V = $\frac{6}{6}$, L V = $\frac{6}{36}$, with marked contraction of the field of vision, and decrease in the light sense of the entire retina. The disk was pale and the vessels narrow.

GALLEMAERTS (506) describes a case of amblyopia from sulphuret of carbon poisoning. The patient was 18½ years old, and his occupation was dipping caoutchouc in sulphuret of carbon in a factory. There had been no abuse of tobacco or alcohol. Besides the amblyopia there were other symptoms, such as muscular weakness, cramps in the calves of the legs, arms, and abdomen, and poor hearing. Central scotoma for all colors, and V = fingers at 45 *cm*, both. After the patient gave up his occupation the vision improved in two months to R = $\frac{1}{20}$, L = $\frac{1}{4}$. MARCKWORT.

HIRSCHBERG (508) in 98 eyes of individuals between sixty and eighty found but 22 normal. For the most part there were slight changes in the retina, attributable to arterial disease.

KOENIG (509) described three old and one new case of thrombosis of the retinal arteries. Ophthalmoscopically the vessels affected most frequently show the blood current interrupted by white masses at intervals. At times the coagulæ are not white, but yellow. The vessels may also be entirely obliterated.

MARCKWORT.

LAWFORD'S (510) patient, a man æt. seventy-one, with dilated heart, partially lost his sight from embolism of one of the branches of the lower division of the central artery, the position of the embolus being visible. WERNER.

WERNER (511) gives an illustration of a horse-shoe-shaped vein dipping into the disk at both its extremities. WERNER.

FERNANDEZ (512) describes a case of traumatic detachment of the retina downward and outward of six weeks' standing, cured by puncture of the ocular membranes 5 *mm* behind the cornea. The puncture gave exit to a clear fluid. Two months later a bluish white patch, due to partial atrophy of the choroid, could be seen with the ophthalmoscope at the point where the detachment had been. The author attributes this exceptional result to the fact that the detachment was traumatic, the eye being previously healthy.

XIX.—OPTIC NERVE.

513. HANSEN, M. A case of acute retrobulbar neuritis. *Med. Record*, Nov. 8, 1890.

HANSEN reports a case of a woman who, after an attack of influenza, was taken with severe headache, which gradually increased in violence. There was slight fever, but no delirium or unconsciousness. After some days V began to fail, and in three days was entirely gone. Pupils dilated ad maximum, and did not respond to light. The ophthalmoscope showed signs very like embolism of the central artery of the retina; veins large, arteries very small, and disks whitish. No heart disease, and urine normal. The retinal arteries gradually filled up, and vision returned to $\frac{5}{200}$, at which it remained stationary. The disk still appears whitish. BURNETT.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

514. DOYNE, R. Perforation of cornea and lens by a chip of steel without subsequent opacity of the lens. *Trans. Ophth. Soc. Unit. Kingd.*, vol x., p. 195.

515. GAZIS. Corps étranger enclavé entre l'iris et la cornée. *Rec. d'opht.*, 1890, p. 638.

516. GUNN, M. Persistent mydriasis following comparatively slight injury. Corresponding cicatrices in cornea and anterior capsule. Advanced globular degeneration of lens. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 196.

517. HUYSMANN, A. Paralysis n. laryngei (securrentis inf.) completa et ptosis sympatico-paralytica (Horneri) traumatica. *Weekblad van het Nederlandsch Tydschrift voor Geneeskunde*, 1890, ii., p. 790.

518. SCHLEICH. A case of subretinal cysticercus, with remarks on the frequency of cysticercus in Wurtemberg. *Med. Correspondenzblatt des Würtemberg. ärztl. Landes-Vereins*, vol. lx., No. 22, p. 169.

519. SNELL, S. The employment of the electro-magnet in ophthalmic surgery. Additional cases and remarks. *Brit. Med. Jour.*, Nov., 1890.

GAZIS (515) reports a case in which a piece of iron, 4 *mm* long and 2 *mm* wide, flew into the eye and remained fixed between the cornea and the iris. An attempt at extraction with forceps was unsuccessful. Even after an iridectomy the foreign body could not be removed with forceps, but when an electro-magnet was introduced the iron was easily extracted. A short time previously the author extracted a piece of iron from the anterior chamber in a similar manner. The foreign body was encapsuled in an exudation, was not visible, and its presence was doubted. When the magnet was brought near the eye the patient felt a movement, and the exudation surrounding the foreign body moved freely, following the motion of the magnet outside the cornea. The diagnosis being thus made, the anterior chamber was opened and the bit of iron easily removed. MARCKWORT.

GUNN (516) observed a case of permanent mydriasis after a comparatively trivial injury. There were cicatrices in the cornea and lens corresponding in position. A number of myelin globules were seen in the anterior capsule of the lens. There was no rupture of the sphincter iridis, since eserine caused an energetic reaction. WERNER.

HUYSMANN'S (517) patient was wounded in the neck with a small knife. The wound healed rapidly, but soon a peculiar hoarseness was observed, with slight ptosis of the right eyelid and

a red color in the right cheek. The right pupil was round, but smaller than the left, and reacted more sluggishly. No further changes. The hoarseness depended upon a total immobility of the right half of the larynx. The sensibility of the mucous membrane was not altered. In the neck there was a cicatrix 18 *mm* long. Huysmann's diagnosis was: Laesio n. vagi dextr. et n. sympathici cervicalis (gangl. medii?).

SCHLEICH (518) found that in twenty years only four cases of intraocular and two of subconjunctival cysticercus had been observed in Wurtemberg. Tænia medio-cannellata is seen more frequently than T. solium.

SNELL (519) reports thirty-seven new cases in which he has employed the electro-magnet. In nine cases the foreign body was in the anterior chamber, in twelve cases in the vitreous. Of the rest one was in the tear sac and the others in the conjunctiva, cornea, and sclera. Cases in which the foreign body lies in the anterior chamber are the most favorable for restoration of vision, those on the contrary in which the foreign body is in the vitreous are the most unfavorable. WERNER.

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

520. ABERCROMBIE, J., and GUNN. A case of uniocular proptosis with double papillitis and intracranial bruit. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 243.

521. ANDERSON, J. Homonymous hemianopsia; recovery; subsequent death and necropsy. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 243.

522. BRAUNSTEIN. Ocular affections with influenza. *Wjestnik Ophth.*, Nov.–Dec., 1890.

523. DOYNE, R. Recovery from hemianopsia; subsequent necropsy. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 235.

524. GAZIS. Des complications oculaires à la suite de l'influenza. *Red. d'opht.*, 1890, p. 586.

525. HENSCHEN, S. Clinical and anatomical contributions to the pathology of the brain. Part I., with 36 plates and 3 diagrams. Upsala, 1890.

526. TILLEY, R. Report of a case of homonymous hemianopsia with a wound in the occipito-parietal region. *Jour. of the Amer. Med. Assoc.*, Oct. 11, 1890.

527. HARDIE and C. A. WOOD. Two cases of nasal hydror-

rhœa with a report on the eye-symptoms. *New York Med. Jour.*, Sept. 6, 1890.

528. KOENIG. De l'artério sclerose et des affections oculaires qui en dépendent. *Thèse de Paris*, 1890.

529. LIEBREICH. Remarkable cases of Basedow's disease, observed in Prof. Schöler's clinic. *Zehender's klin. Monatsbl. f. Augenheilk.*, 1890, p. 492.

530. MORTON, A. S. Alexia with right-sided hemianopsia. *Trans. Ophth. Soc. Unit. Kingd.*, vol. x., p. 247.

531. NOYES, H., and DANA, L. A case of tumors of the ventricle with left anophthalmus. *Med. Record*, July 16, 1890.

532. PANAS. Rapport de l'ophtalmologia avec la médecine générale. *Rec. d'opht.*, 1890, p. 633.

533. DE SCHWEINITZ, G. The ocular complications of malaria. *Med. News*, June 7, 1890.

ABERCROMBIE and GUNN (520) describe the case of a child, æt. two and one-half years, who had an intracranial bruit, heard all over the head; there was double optic neuritis with proptosis of left eye. Some history of a headache; no injury. In six months the child was almost blind, disks white. Proptosis unaltered. WERNER.

In ANDERSON'S (521) case, a man, æt. forty-one, there was failure of vision with severe frontal headache, loss of flesh. He was found to have right hemianopsia, the fields varied a little from time to time, he subsequently had vomiting, and a transitory attack of left hemiplegia. *Post mortem* a glioma of the left angular gyrus was discovered. Nothing to account for the hemiplegia. WERNER.

BRAUNSTEIN (522) reports the following cases: I. A girl, æt. fourteen, previously strong and healthy, had an attack of typical epidemic influenza with high temperature, severe headache, and vomiting. Soon after her recovery she noticed diplopia, strabismus and giddiness. A month after the influenza the tendon reflexes in the upper extremities were weak, the patellar reflex altogether wanting. Sensibility of skin of right leg diminished. Pupils dilated, and reacting sluggishly. Bilateral abducens paralysis (right more marked). Severe neuro-retinitis, both. V = $\frac{20}{100}$ O. U. Diagnosis: neuritis multiplex. Treatment: hot foot-baths, cantharides to the neck, internally infus. jaborandi with sod. salicyl. Rapid marked improvement. II. A healthy man had an

attack of typical influenza and noticed on the fifth day, when the temperature was lower, that his vision was poor, and twelve hours later he was entirely blind. Examination some days later showed complete blindness in both eyes, disks of normal color, perhaps somewhat hazy, and the retina slightly hazy. Arteries small, veins tortuous and dilated. Temperature normal. Pulse 130; otherwise normal condition. No results from treatment. Diagnosis: probably retrobulbar neuritis (ischæmia retinæ, in consequence of weakness of the heart not excluded). III. In a tabic patient, æt. fifty-three, a serous iritis developed in the left eye, one week after an attack of influenza. It ran its natural course. IV. A severe suppurative choroiditis in one eye in the course of a typical influenza. HIRSCHMANN.

DOYNE'S (523) patient, a man, æt. seventy, suddenly became confused and was found to have right hemianopsia, the vision and fields altered from time to time, the right halves recovering, and the left lower quarters were at one time lost. He subsequently developed paresis of the left arm and right side of face, and aphasia. He died unconscious. Left hemisphere of brain, small patch of softening at upper end of perpendicular fissure. Right cerebral hemisphere, three patches of softening, one cortical near tip of occipital lobe, the two others also in occipital lobe.

WERNER.

GAZIS (524) describes the complications of the influenza that were observed in Galezowski's clinic. They were as follows: Choked disk, herpes corneæ, conjunctivitis, and episcleritis. Gazis speaks in detail of the choked disk and reports a new case of that sort. MARCKWORT.

HENSCHEN'S (525) work, which is of great value respecting the course of the visual tracts in the brain and the clinical symptoms of hemiopia, cannot satisfactorily be reviewed here on account of its size. The microscopic illustrations were prepared from not less than ten thousand preparations. A detailed review may be found in the *Berliner klin. Wochenschr.*, 1890, p. 1096.

TILLEY (526) reports the case of a young man with a typical left homonymous hemianopsia which seemed to follow a blow on the back part of the head. The pupils contracted when light was thrown on the blind part of the retina. Syphilis seemed to be excluded, and otherwise the patient appeared in good health.

BURNETT.

The eye symptoms noted by HARDIE and WOOD (527) in the

cases of nasal hydrorrhœa were dimness of vision and a concentric narrowing of the visual fields. There was no color-blindness. The disks showed strips of atrophy at the periphery of the nerve entrance. Fundus otherwise normal. BURNETT.

As complications of Basedow's disease, LIEBREICH (529) observed an ulceration of the cornea in both eyes, a constant peculiar twitching of the lids, twice an ophthalmoplegia externa, and once a paresis of the right abducens.

In the case of NOYES and DANA (531) the man had lost the left eye by enucleation in 1873, fifteen years before he was seen by them. Besides the other general symptoms of brain tumor, those referable to the eyes, were: paralysis of sixth nerve on the right side, branches of the third to the levator palp., the sixth on the left side, irritation of fourth and the branches to sup. and inf. recti, causing rotating and vertical nystagmus. A section of the decussation of the optic nerves showed a band of degenerating tissue crossing from the upper part of the left nerve to the lower part of the right tract; there was no degenerated band in the left tract, but there was in the right, seeming to show a total decussation of the nerve fibres. The eye symptoms also seem to indicate a larger nucleus for the levator palp. than has been supposed to exist. The ciliary muscle and iris were not involved. BURNETT.

PANAS (532) reports the case of a boy, æt. seven, in whose right eye there had been severe hemorrhage into the vitreous; in the left there was seen the disease which Manz had described as retinitis proliferans. There was an organized plaque in the external portion of the disk and in front of this opacities in the vitreous. These plaques were of hemorrhagic origin. The ocular hemorrhages, according to Panas, were induced by a vaso-motor reflex proceeding from the intestinal canal. The patient suffered with recurring diarrhœa. MARCKWORT.

DE SCHWEINITZ (533) gives the histories of several cases, and cites the literature of the whole subject of the ocular complications of malaria, and concludes: (1) that there is an intermittent ophthalmia, malarial in its nature; (2) that there is a true and distinct malarial keratitis; (3) that various functional disturbances, such as amblyopia, paresis of accommodation, alterations in the visual field, hemianopsia, and night blindness, can be malarial in origin; and (4) that there may be gross changes from the same cause, such as neuritis, atrophy of the nerve, and hemorrhages. BURNETT.

REPORT ON THE OPHTHALMOLOGICAL SESSION OF THE SPANISH MEDICAL CONGRESS, HELD IN VALENCIA, JULY, 1891.

Treatment of Granulations by Means of the "brossage."—Dr. OSIO, of Madrid, dwelt on the tenacity of this disease and the inefficiency of almost every treatment, some of them being even dangerous, such as the employment of *jequirity* and excision of the conjunctival cul-de-sac. He generally resorts to the "*brossage*" with a hard tooth-brush, after having inverted the eyelids and scarified the conjunctiva. He performs the inversion of the eyelids with a forceps especially devised for this purpose. The brushing, when performed after scarification, has given a most prompt success.

Dr. LLORET, of Valencia, is of the same opinion, and pronounces himself against the excision of the cul-de-sac suggested by Galezowski, which he believes is apt to produce entropion by cicatricial retraction.

Dr. SANTOS FERNANDEZ, of Havana, said that all the methods of treatment suggested against granulations are deficient because they are unphysiological; they succeeded in removing granulations by destruction of the conjunctival epithelium, this being the ultimate result of granulations when left alone. He agrees that the excision of the cul-de-sac is a defective method, but affirms that it is free of danger; he has considerable experience in it. Knapp's treatment by pressing out the trachomatous substance with special roller forceps is the only conservative treatment, but he has not yet formed an opinion as to its efficiency.[1] In the meantime he accepts brossage, notwithstanding the necessary destruction of epithelium, for this method is the only one that will certainly extinguish granulations.

Dr. AGUILAR BLANCH believes that "excision" is free of danger, and that there is no single treatment that can be resorted to

[1] I have operated on fifty-six cases. The results are most satisfactory. No scars where there were none before. Treatment in one sitting. No relapses. —Sept. 8, 1891.—H. KNAPP.

in every case; the treatment must be different, according to the nature of the disease.

Loss of Sight in Yellow-Fever.—Dr. SANTOS FERNANDEZ read a paper on this most rare complication; he had only heard of three cases; one he had observed himself, and the other two he had been informed of by two colleagues. Ophthalmoscopic examination was performed in two cases, and it was proved that the eye was free from organic lesions. In the case which he observed himself, anuria and violent delirium were present; all three patients died within the first week.

Dr. OSIO, by comparison with other infectious diseases, thinks that the loss of sight is due to anuria.

Ocular Lepra.—Dr. AGUILAR BLANCH: In ocular lepra the eyelids are affected with anæsthesia, nodules, naderosis, and ectropion; entropion is of rare occurrence; the conjunctiva is also involved in the same processes; the cornea is also inflamed late; the sclerotic and iris are not spared; the iris is generally subject to chronic inflammation.

Dr. SANTOS FERNANDEZ agrees with the reader, for he has been able to confirm every fact in the Havana Leper Hospital. The same is the case with his colleague, Dr. Lopez, author of the paper mentioned by Dr. Aguilar; in a doubtful case diagnosis was established by bacteriological examination.

New Method for the Correction of Strabismus.—Dr. AGUILAR isolates the muscle and the capsule from the sclerotic; he does not divide the muscle nor does he employ any suture; the tendon is afterwards shortened by suture. He exhibited a new instrument for the measurement of the degree of retraction.

Dr. SANTOS FERNANDEZ says that if the method would not have proved successful in the hands of the author he would for theoretical reasons have deemed it inefficient.

Dr. OSIO thinks that instruments for the measurement of the muscular retraction are poor in practical results.

Inheritance of Cataract.—Dr. AGUILAR speaks of a family whose members are strongly predisposed to cataracts, and shows the cases arranged in a table.

Dr. SANTOS FERNANDEZ has observed hereditary cataracts in a branch of his own family, and thinks that in the explanation of these facts a place must be left for mere coincidence. Thousands of cases are treated that show no family predisposition.

Post-Traumatic Cataract.—Dr. VIRCIANO reports a number of these cases; he endeavors to explain the fact by the opacity of

the crystalline lens, and asks whether these cataracts should be operated on or not.

Dr. SANTOS FERNANDEZ says that traumatisms are sometimes overlooked by the patients; that cataracts are more satisfactorily treated in old than in the young persons.

Method for the Removal of Staphyloma.—Dr. OSIO simply cuts off the staphyloma, passing his knife before the iris, and finishes the removal of the opaque cornea with the scissors; after this he applies a compressive bandage; the presence of the iris largely contributes to the normal appearace of the eye.

Treatment of Short-Sightedness by Extraction of the Crystalline Lens.—Dr. SANTOS FERNANDEZ has observed the visual power increased in myopes after the operation for cataracts; he consequently thinks this method can be resorted to in those cases in which short-sightedness has so much progressed as to constitute a serious danger for the retina. He has recently seen two successful cases in Dr. Abadie's ophthalmological clinic. He thinks that discission should be performed in the young and extraction in the old.

Dr. AGUILAR agrees with Dr. Santos Fernandez, but thinks that before transparent crystalline lenses are to be extracted more evidence is needed.

Dr. BAYARRI relates a case which strongly confirms the opinions of Dr. Santos Fernandez.

Dr. OSIO does not think that myopes should be treated by extraction, because this would expose them to a useless danger.

Dr. SANTOS FERNANDEZ says that he only proposes the method for those cases over 20 dioptries, as the length of the anterior diameter is shortened by the operation, thus lessening the dangers of the most advanced degrees of progressive myopia.

Ocular Auto-Infection.—Dr. SANTOS FERNANDEZ reported two cases in which suppurative irido-cyclitis had taken place several days after the operation. In one case the infection was attributed to a focus of suppuration of the face, probably due to the irritative action of bichloride; in the second case, as no cause was found responsible for the infection, it was deemed endogenous, and probably due to the weak resisting power of the patient.

Dr. AGUILAR admits auto-infection only in those cases where no filtering surface is left.

Dr. CANDELA said that he admits auto-infection, although the cases are not always clearly understood, because he has observed and thoroughly studied them in obstetrics.

CORRIGENDA.

I.—In Dr. A. Percival's paper on Prismospheres, in this volume, the following errors should be corrected.

P. 195, line 6 from bottom, *for* "the focal distance of the lens; D represents," *read* "the focal distance of the lens in millimetres; if D represents."

P. 199, line 14, *for* "divergence is required, the decentration must be inwards," *read* "convergence must be maintained, the decentration must be outwards."

P. 199, line 19, *for* "outwards $2 \chi 2.661$ *mm*," *read* "inwards 2×2.661 *mm*.

P. 199, footnote, *for* "$10 \tan 7^\circ = .174551 = \tan 9^\circ - 54'.0767$," *read* "$10 \tan 1^\circ = .174551 = \tan 9^\circ\ 54'.0767$."

P. 200, line 16, *for* "object," *read* "defect."

P. 202, after the first paragraph insert the fourth paragraph on p. 203. After the second paragraph insert the third paragraph on p. 203.

P. 203, line 5 from bottom, add after "prismospheres," "in suitable cases."

P. 204, column 1 D, 4th line *for* "31.120" *read* "31.121."

P. 205, line 5 from bottom, *for* "presented," *read* "presumed."

P. 205, last line, *for* "junction," *read* "function."

P. 206, column — 4 D, line 8, *for* "— 2.6," *read* "2.6."

P. 206, column — 7 D, line 3, *for* "26.6," *read* "26.7."

P. 206, last line, *for* "junction," *read* "function."

II.—In my paper "On the Proposed Methods of Numbering Prisms" (ARCHIVES OF OPHTHALMOLOGY, XX., 3), the words "and which has been copied in Prentice's article on 'The Prism-Dioptry' (ARCH. OF OPHTHALM., XIX., 2 and 3, p. 128)" should be expunged. The reference should have been to Percival's paper in the same number, p. 257, of the ARCHIVES.

In Prentice's paper, the formula given, namely:

$$R = \sqrt{P^2 + Q^2},$$

in which R, P, and Q represent prism-dioptries and therefore tangents, is practically the same as the amended formula:

$$\text{Tan. } R = \sqrt{\text{Tan.}^2 H + \text{Tan.}^2 V},$$

given on page 326 in my article.

A. DUANE, M.D.

ANNOUNCEMENT.

OWING to the steady increase of important contributions (freely illustrated by numerous wood engravings and superb lithographic and colored plates) which have been accepted for publication in the ARCHIVES OF OTOLOGY, the Publishers find themselves compelled to raise the annual subscription price from $3 to $4. The latter is the price at which the German edition, though costing less money to produce, has been issued ever since the separation of the ARCHIVES OF OPHTHALMOLOGY AND OTOLOGY into two independent periodicals. No reduction can be afforded to subscribers taking the two journals, ARCHIVES OF OPHTHALMOLOGY, and ARCHIVES OF OTOLOGY, and the subscription for the two will be hereafter $9 a year. The increase in price will go into effect January, 1892.

G. P. PUTNAM'S SONS, PUBLISHERS.

SEPTEMBER, 1891.

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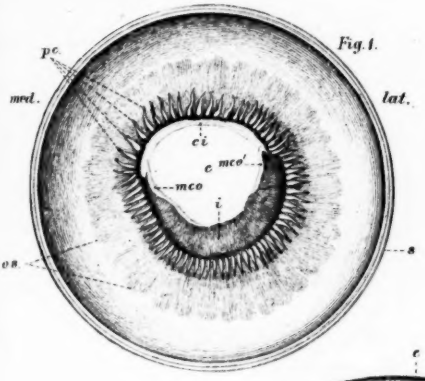

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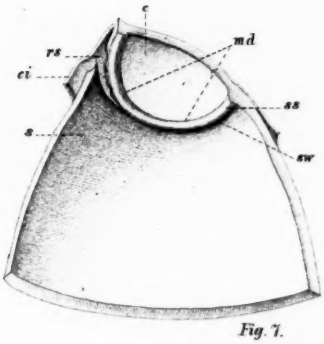

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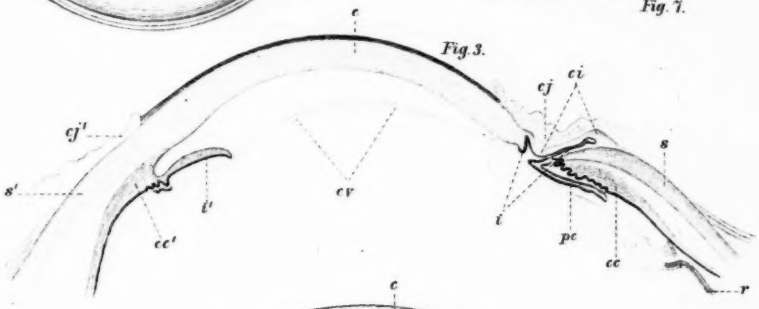

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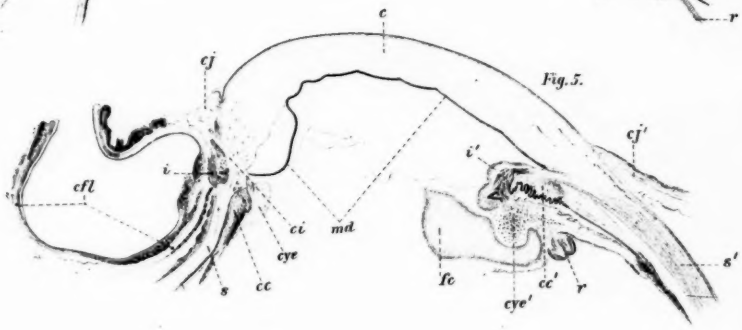

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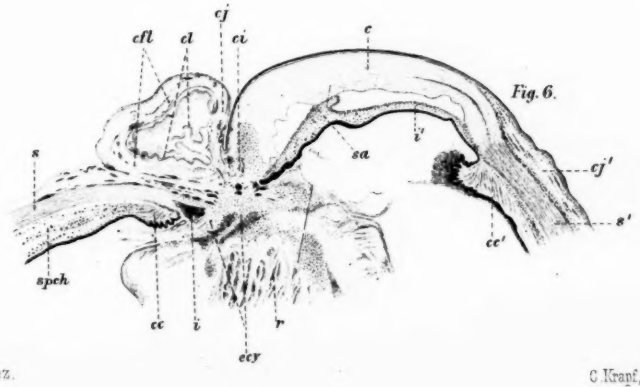

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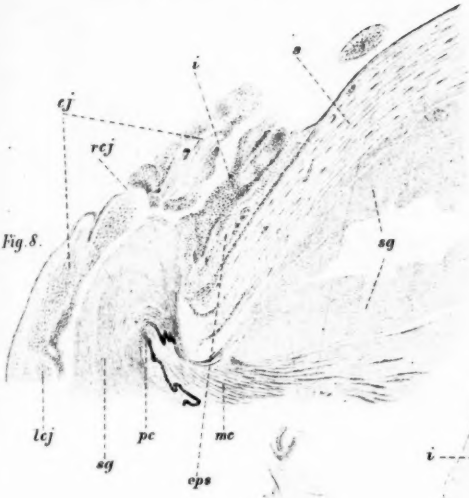

Fig. 2.

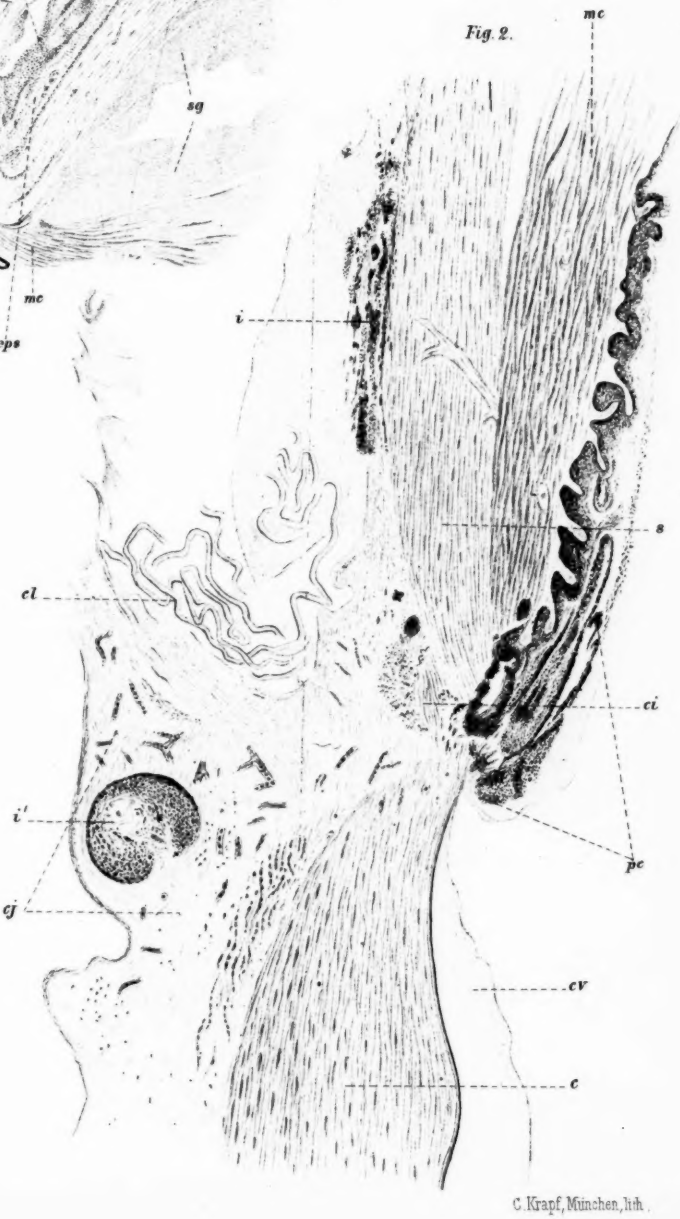

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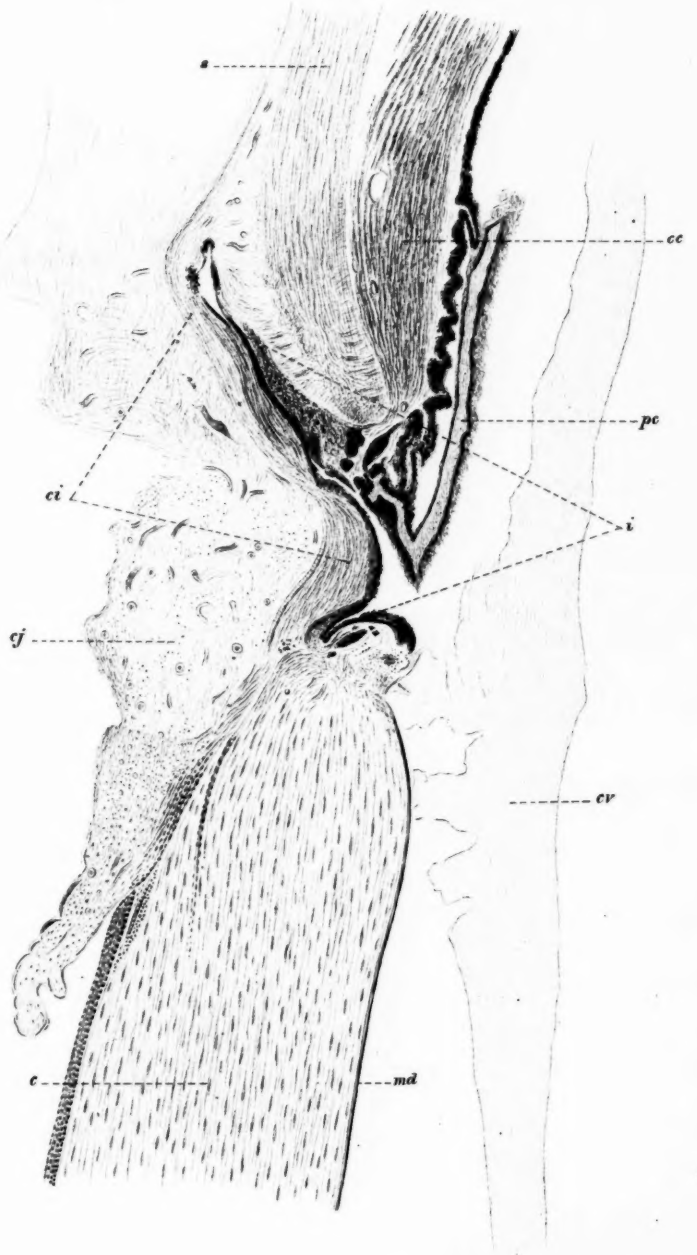
Fig. 4.



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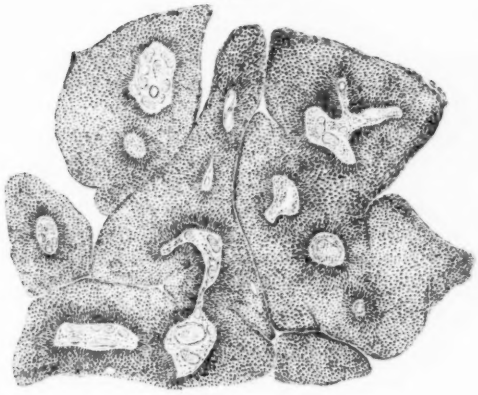

Fig. 2.

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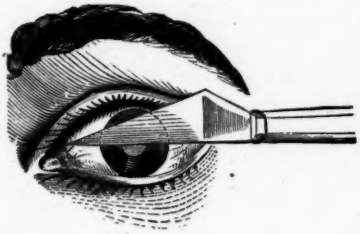

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